Consideration for preliminary adoption of amendments to 312 IAC 9-10-9 that govern wild animal rehabilitation permits; Administrative Cause No. 10-015D

Wild animal rehabilitation permits allow qualified individuals to take in wild animals (including mammals, birds, reptiles, and amphibians) for the purpose of caring for the animal until it can be released back into the wild. Many of the animals are orphaned young, but other animals may be sick or injured adults. While wild animal rehabilitators have good intentions, the DNR has had to take legal action on multiple permit holders within the past few years. Violations have included off-site rehabilitation of wild animals by assistants, the failure to release animals, not releasing animals within the timeframe required, filthy and unsanitary rehabilitation facilities that have even warranted action by county health departments, and attempts to transfer of these animals to a game breeder license (which allows the sale of these animals). These issues, as well as others, necessitated the forming of a Wild Animal Rehabilitation Advisory Group to discuss modifying the existing rehabilitation permit rule.

The majority of these rule changes are a result of the work of an advisory group that consisted of the following members: Kathy Hershey (licensed rehabilitator), Kim Hoover (licensed rehabilitator), Ron Johnson (DNR Conservation Officer), Tim Julien (Nuisance Wildlife Control Operator's Association), Dr. Larry McAfee DVM (licensed veterinarian and licensed rehabilitator), Sandra Norman DVM (Indiana Board of Animal Health), Patti Reynolds (licensed rehabilitator), Dean Zimmerman (DNR wildlife biologist), and Linnea Petercheff (DNR, Division of Fish and Wildlife Operations Staff Specialist). The DNR also took input from other rehabilitators who attended the meetings, but were not part of the advisory group. A letter was mailed to all licensed rehabilitators in November of 2009 asking for their input, and many changes were made as a result of their comments. The advisory group wanted the majority of these changes, including the testing requirement for new rehabilitators.

The majority of the proposed changes are summarized as follows:

1. <u>New Requirements for becoming a licensed rehabilitator</u>
    - Require new applicants to pass a test and those who have had a permit for less than 10 years and taken in less than 12 animals for rehabilitation each year *(recommended by the Wildlife Rehabilitation Advisory Group)*
    - Clarify who can serve as a reference or provide training *(removes current requirements that are not relevant to wildlife rehabilitation and includes only those that have actual experience or training in caring for/handling wild animals)*
    - Mandatory reference materials *(the DNR will have a list that will be distributed to all applicants; references are being put together from rehabilitators that have been licensed for over 15 years)*
    - Inspection by a conservation officer for all new applicants *(consistent with other permits issued by the DNR and ensures that the applicant has appropriate facilities and understands the requirements)*

2. <u>Housing Requirements</u>
    - Can't be housed with domestic animals and other animals without written approval from DNR *(disease issues)*
    - Can't be used for public display *(currently not allowed but this makes it clear; wild animals undergoing rehabilitation should not be subjected to other people that can impede their rehabilitation; also prohibited for the rehabilitation of migratory birds in 50 CFR 21.)*
    - Must keep enclosures clean and in sanitary conditions *(this has become a problem in recent years with several rehabilitators who cannot say 'no' to people and take in more animals than they can house and care for properly; county health departments have been involved in 2 cases)*

- Prevent human contact as much as possible *(to prevent habituation to humans and help prevent the animal from viewing humans as their source of food – especially raccoons and deer)*

3. Clarifies requirements for assistants
   - Limits use of assistants and clarifies what they can do *(clarifies current requirements; many assistants are rehabilitating wild animals at their own facility with little or no supervision)*

4. Release requirements
   - Must release within a specific time period (usually 180 days) unless approval is given due to time of year and extensive injuries *(see justification in the attachment)*
   - Must have permission from the landowner where animals are released *(private property right)*
   - Restrictions on where animals can be released *(see justification in the attachment)*

5. Non-releasable animals
   - Clarifies that non-releasable animals can be kept as foster parents or as education animals, but not as pets
   - Limits the number of non-releasable animals a rehabilitator can keep (*currently no limits, but a wild animal possession permit is already required for mammals other than white-tailed deer and an education permit is required for non-releasable birds and for mammals other than deer used in education programs*)
   - Requires non-releasable white-tailed deer to be euthanized (*currently required in non-rule policy*)

6. Clarifies disposition of dead animals
   - Clarifies the legal use of dead animals – mounting for display purposes, donate to educational institutions, etc.
   - Cannot be sold or bartered *(already not allowed by law in IC 14-22-6-3 but clarified by putting it in this rule)*
   - Require humane methods of euthanasia *(these will be the methods recommended by the National Wildlife Rehabilitator's Association)*

7. Rehabilitation of certain species (*see justification in the attachment)*

   - Require all diseased wild animals, except for migratory birds, to be euthanized
   - Require all severely injured wild animals, except for migratory birds and turtles with shell fractures, to be euthanized unless determined by a licensed veterinarian to be treatable and released within 180 days from the date in which the animal was originally obtained by the licensed rehabilitator
   - All mammals, reptiles, and amphibians must be released in the county where the animal was originally found or obtained unless the origin is unknown

Please see the attached justification regarding the release requirements for raccoons, foxes, coyotes, skunks, and opossums; and the purpose for the requirement to release within 180 days.

The DNR is recommending preliminary adoption of these changes.

TITLE 312 NATURAL RESOURCES COMMISSION

Proposed Rule
LSA Document #10-

DIGEST

Amends 312 IAC 9-10-9 governing the wild animal rehabilitation permit. Effective thirty (30) days after filing with the Publisher.

312 IAC 9-10-9

SECTION 1. 312 IAC 9-10-9 IS AMENDED TO READ AS FOLLOWS:

312 IAC 9-10-9 Wild animal rehabilitation permit
Authority: IC 14-22-2-6; ~~IC 14-22-11-12~~
Affected:IC 4-21.5; IC 14-22

Sec. 9. (a) This section governs a permit to possess a wild animal for rehabilitation. The permit is required ~~for~~ **to temporarily possess any wild animal that is** a mammal, bird, reptile, or amphibian **for rehabilitation purposes** and is available only to an individual who is a resident of Indiana. ~~A white-tailed deer must not be possessed under this section for more than one hundred eighty (180) days unless a conservation officer inspects the animal and determines an extended period may be reasonably expected to result in its rehabilitation.~~

(b) **An individual may, without a permit, take possession of a sick, injured, or orphaned wild animal and transport it to an individual with a valid permit under this section within twenty-four (24) hours.**

~~(b)~~ **(c)** An application for a permit under this section shall be completed on a departmental form and must ~~establish~~ **provide** the following:
(1) The applicant has ~~rehabilitation experience and a knowledge of wildlife rehabilitation techniques. The required~~ **one year of** experience ~~and knowledge may be met by~~ **with** one (1) of the following **types of facilities that works with the species to be rehabilitated**~~:~~
~~(A) A bachelor of science degree in a wildlife related field.~~
~~(B) At least one (1) year of experience with a:~~
~~(i)~~ **(A) licensed** veterinarian **who has a wild animal rehabilitation permit;**
~~(ii)~~ **(B) zoological park;**
~~(iii)~~ **(C)** university animal clinic;
~~(iv) county animal shelter; or~~
~~(v)~~ **(D)** licensed ~~rehabilitation clinic~~ **wild animal rehabilitator who has had a permit for at least three (3) years.**
~~(C) Possession for at least two (2) years of another permit under this section.~~
~~(D) Other knowledge and background, including the completion of rehabilitation workshops and seminars, if found by the division director to qualify the applicant.~~
Documentation of the experience with a licensed veterinarian, university animal clinic, zoological park, or licensed wild animal rehabilitator must be submitted with the application.
(2) The name and address of a veterinarian willing to assist the applicant with the rehabilitation of wild animals. The veterinarian shall sign the application and attest to having experience in the care ~~and rehabilitation~~ of **the species of** wild ~~mammals and birds~~ **animals to be rehabilitated**. If the applicant is a veterinarian, the signature of another veterinarian is not required.

(3) A listing of the wildlife rehabilitation reference books in possession of the applicant. **The individual must have at least one (1) reference book that:**

(A) includes information pertaining to each species listed on the application form, and

(B) is on the list approved by the department.

(4) The names, addresses, and telephone numbers of any other individuals who will assist the applicant ~~Assistants must possess sufficient experience and adequate facilities to tend the species in their care and be authorized in writing by the permit holder to provide care for that species of animal in their own facility~~.

(5) The species **or type of animals** that will be accepted for rehabilitation.

(6) A description of the rehabilitation facilities, equipment, and supplies. The description shall include the following:

(A) Cages.

(B) Intensive care units.

(C) Aviaries.

(D) ~~Falconry equipment.~~

~~(E)~~ ~~Medical diagnostic equipment~~.

~~(F)~~ Medical supplies.

~~(G)~~ ~~Food sources~~.

~~(H)~~ **(E)** Other items to be utilized in the rehabilitation process.

A cage description shall provide its internal dimensions and shall specify the materials used for flooring, walls, and perches. ~~The applicant shall list what species will be housed in the various enclosures and the purpose for each enclosure, for example, convalescing, training, or quarantine~~.

(7) The applicant is at least eighteen (18) years old.

~~(c)~~ **(d) Before a permit may be issued the applicant must correctly answer at least eighty percent (80%) of the questions on a written examination supervised and administered by the department covering basic biology, care of wild animals and the laws relating to wild animal rehabilitation.**

(1) An individual who fails to correctly answer at least eighty percent (80%) of the questions on the examination may retake the examination no more than two (2) additional times within ninety (90) days, but not again within one hundred eighty (180) days after the third failure.

(2) Exempted from this subsection is an individual who has had a permit under this section for ten (10) years and taken in at least twelve (12) wild animals each year for rehabilitation. However, all individuals who have a permit under this subsection must meet the requirements in subsection (e).

(e) A permit holder who has satisfied subsection (d) must, within three (3) years of being issued a permit, either:

(A) satisfy the same requirements as are set forth in subsection (d) on another examination; or

(B) complete eight (8) hours of continuing education as approved by the division, including courses that will be sponsored by the department, National Wildlife Rehabilitator's Association or International Wildlife Rehabilitation Council.

(f) A conservation officer must inspect the cages and any other enclosures where wild animals will be housed before a permit may be issued to a new applicant. A conservation officer may inspect the enclosures, wild animals, and any records relative to a permit issued under this section at any reasonable hour.

(g) An amended application **or written request** must be filed with the division if there is a ~~material~~ change to the ~~information~~ **address of the applicant, the name of the assisting veterinarian, the identity of assistants or the addition of species of wild animals that was** provided in the original application. ~~The amended application must include the name, address, and telephone number of any additional person who would assist the permit holder.~~

~~(d)~~ **(h)** The permit holder must file an application by January ~~15~~ **31** of each year in order to renew the permit. The annual report required under subsection ~~(i)~~ **(m)** must accompany the renewal application. The signature of a veterinarian is not required for a renewal application.

~~(e)~~ **(i)** The issuance of a permit under this section does not relieve an individual from any requirement for a federal permit. If the terms of a federal permit and the permit issued under this section differ, the more restrictive terms prevail.

~~(f)~~ **(j) Public exhibition or display of any** ~~A~~ wild animal possessed pursuant to a permit issued under this section ~~must not be displayed or placed in physical contact with the public, except according to the terms of an educational permit issued under section 9.5 of this rule.~~ **is prohibited. Only assistants and other individuals dropping off an animal for rehabilitation at the permit holder's facility may view or have contact with the wild animal unless specifically authorized in writing by the department. Electronic viewing and photographs are allowed.**

~~(g)~~ **(k)** A permit holder must maintain facilities for the retention of a wild animal possessed under this section in a sanitary condition **as follows** and ~~to~~ conform with any other conditions specified by the permit~~.~~**:**

(1) Cages must be cleaned daily using non-irritating methods unless medical treatment necessitates otherwise.

(2) The permit holder and any assistants listed on the permit must observe and provide care for wild animals at least once daily unless otherwise specified by the permit.

(3) Wild animals must be kept in enclosures and in an environment that minimizes human contact at all times to prevent imprinting and bonding to humans. The permit holder or designated assistants should interact with the wild animal only to the extent necessary to provide adequate care and treatment.

(4) Wild animals must not be allowed to come into contact with any individuals other than a permit holder, assistant listed on a rehabilitation permit, licensed veterinarian, animal control officer, law enforcement officer, or authorized department employee.

(5) Wild animals must not be housed in a cage that would allow physical contact of the animals undergoing rehabilitation with domestic or companion animals or animals kept under any other license or permit unless authorized in writing by the department.

(6) If suspected of having an infectious disease, wild animals must be quarantined in areas designated for that purpose.

(7) Wild animals must be kept separated from human living quarters and activities unless intensive care is required, but unweaned wild animals may be housed in human living quarters that are away from human activities.

(8) Wild animals must be housed in enclosures that:

(A) are structurally sound;

(B) are of sufficient strength for the species involved;

(C) are maintained in good repair to prevent escape or injury to wild animals being rehabilitated;

(D) are constructed to allow sufficient space for individual posture and social movements, unless medical treatment necessitates restricted mobility,

(E) are secured when unattended;

(F) have protective devices at entrances and exits to prevent escapes if kept outdoors and if needed to prevent injuries to human or the animal's health;

(G) have ambient temperatures that are appropriate for the species located within the enclosure;

(H) have adequate ventilation by means of windows, doors, vents, fans or air

conditioning to protect wild animal health and to minimize drafts, odors and condensation;

(I) maintain adequate lighting by artificial or natural means that is cycled for appropriate photoperiod, if necessary for the species in possession;

(J) provide adequate shade, weatherproof shelters, nest boxes, perches, and dens to protect wild animals from inclement weather and direct sun if kept outdoors.

(9) No exposed sharp objects, ponds with steeply sloped banks, toxic paints or sealants, or poisonous vegetation may be used in the construction of enclosures.

(10) Enclosures shall have either visual barriers or be separated by distance to restrict a wild animal's view of humans and other species being rehabilitated to reduce inappropriate imprinting, socialization, habituation, or stress, unless a view of humans and other wild animals will not affect the animal's ability to survive in the wild.

(11) Wild animals must be fed as follows:

(A) appropriately and adequately to meet nutritional needs unless medical treatment necessitates restricted food intake.

(B) with food that is palatable, free of contamination, and of sufficient quantity and nutritive value for the species involved, including the use of supplemental vitamins and minerals when necessary for recovery.

(C) with food receptacles that are appropriately sized, easily accessible, and kept sanitary and safe.

(12) Wild animals must be given water as follows:

(A) fresh clean water for drinking shall be provided throughout the day unless medical treatment necessitates restricted water intake

(B) additional water shall be provided for species requiring bathing, swimming, or misting unless medical treatment necessitates restricted water exposure.

(C) with water receptacles that are appropriately sized, easily accessible, kept sanitary as much as possible and safe.

(13) Removal and disposal of wild animal food wastes, feces and urine, bedding, from the enclosure and premises shall be performed daily to maintain sanitary conditions and protect wild animal and human health. Trash, garbage, debris, and carcasses must be removed from the enclosure as soon as they are observed and appropriately disposed of.

(14) Cages, rooms, hard surfaced pens, kennels, runs, equipment, and food and water receptacles shall be sanitized between each adult wild animal use and between litters to prevent disease transmission.

~~(h)~~ **(l)** A permit holder must maintain current records for each wild animal to include the following:
(1) The species and condition of the animal.
(2) The name, address, and telephone number of the donor or other source of the animal.
(3) The date of receipt by the permit holder.
(4) The treatment**, including the identification of and date of administration of any pharmaceutical product or other chemical** provided to the animal while in captivity.
(5) The method and date of disposition of the wild animal.
All records must be maintained at the facility of the permit holder for a minimum of three (3) years.

~~(i)~~ **(m)** The permit holder shall provide ~~an~~ **a signed** annual report to the division by January ~~15~~ **31** of each year. The report shall list the following:
(1) The species and condition of each animal.
(2) The date the animal was received.
(3) The name and ~~address~~ **county** of the donor or other source.
(4) The method, location, and date of disposition of the animal.

The report may be a computerized record signed by the permit holder under oath or affirmation to be a true and accurate account of all wild animals taken in for rehabilitation during that year or on a form provided by the department and must be maintained at the facility of the permit holder for a minimum of three (3) years.

~~(j)~~ **(n)** As soon as a wild animal is capable of fending for itself, the animal ~~shall~~ **must** be released into the wild as ~~directed by a conservation officer. If a wild animal is not capable of fending for itself, a conservation officer must be contacted for instructions concerning its disposition.~~ **follows:**

(1) The wild animal must be released on land only with permission of the landowner.

(2) Mammals must be tagged or marked as directed by the department if given a pharmaceutical product or other chemical for the purpose of tranquilizing or anesthestizing the mammal unless the product administered is labeled as safe for human consumption and the mammal is released after the clearance period.

(3) A bird must be released in the county from which it was originally found except as follows:

(A) A bird that is being released outside the breeding season for that species,

(B) A bird that has been in a rehabilitation facility for ninety (90) calendar days or more, or

(C) a bird that is not in the order Anserifomes, Charadriiformes, Columbiformes, Falconiformes, Galliformes, Gruiformes, or Strigiformes

may be released in appropriate habitat in any county that is within that species' normal range in Indiana.

(4) A reptile or amphibian must be released at the site where found, unless the origin is unknown. If the origin is unknown, the reptile or amphibian may be released in appropriate habitat in the permit holder's county of residence.

(5) For mammals, the following applies:

(A) A mammal must be released in the county from which it was originally found or obtained, unless the origin is unknown. If the origin is unknown, the mammal may be released in appropriate habitat in the permit holder's county of residence.

(B) In addition to the requirements in subsections (n)(1), (n)(2) and (n)(5)(A), raccoons, Virginia opossums, striped skunks, coyotes, and foxes must be released only as follows or be euthanized:

(i) the release site must consist of at least two (2) contiguous acres,

(ii) no more than eight (8) animals per species may be released per two (2) acres of any site per calendar year, and

(iii) no more than twenty-four (24) animals per species may be released on any site by a permit holder in a calendar year.

(C) In addition to the requirements in (n)(1), (n)(2) and (n)(5)(A), white-tailed deer must be released only as follows or be euthanized:

(i) the release site must consist of at least five (5) contiguous acres,

(ii) the release site must be outside city limits, and

(iii) no more than ten (10) white-tailed deer may be released per site per calendar year.

(o) A permit holder may keep a non-releasable wild animal, other than a white-tailed deer, only if approved by the division of fish and wildlife as follows:

(1) for educational purposes under a special purpose educational permit under 312 IAC 9-10-9.5 as follows:

(A) for mammals other than white-tailed deer:

(i) a wild animal possession permit must be obtained under 312 IAC 9-11,

(ii) no more than three (3) mammals per species and six (6) mammals total may be possessed if twelve (12) or fewer total mammal programs are presented to the public each calendar year,

(iii) no more than five (5) mammals per species and no more than twenty (20)

mammals total may be possessed if thirteen (13) or more total mammal programs are presented to the public each calendar year,
(iv) exceptions to these numbers may be granted upon prior written approval from the division based on the number of educational programs presented to the public in the previous year.

(B) For birds:
(i) a special purpose educational permit must be obtained under 312 IAC 9-10-9.5,
(ii) no more than three (3) birds per species may be possessed and six (6) birds total if twelve (12) or fewer programs with each bird are presented to the public each calendar year,
(iii) no more than five (5) birds per species may be possessed and no more than twenty (20) birds total if thirteen (13) or more bird programs with each bird are presented to the public each calendar year,
(iv) exceptions to these numbers may be granted upon prior written approval from the division based on the number of educational programs presented to the public in the previous year.

(C) For reptiles:
(i) a turtle possession permit must be obtained under 312 IAC 9-5-11 to possess an eastern box turtle and a wild animal possession permit must be obtained under 312 IAC 9-11 to possess an endangered species of reptile.
(ii) no more than three (3) reptiles per species and six (6) reptiles total may be possessed if twelve (12) or fewer total reptile programs are presented to the public each calendar year,
(iii) no more than four (4) reptiles per species and no more than twenty (20) reptiles total may be possessed if thirteen (13) or more total reptile programs are presented to the public each calendar year,
(iv) exceptions to these numbers may be granted upon prior written approval from the division based on the number of educational programs presented to the public in the previous year.

(2) for fostering other wild animals only if five (5) mammals or birds of the same species have been reported in at least the two (2) previous years.
(3) wild animals that have been used in educational programs or as foster parents and are no longer capable of being used in that capacity due to age or health may be kept with written permission from the division of fish and wildlife.
(4) wild animals possessed lawfully prior to July 1, 2010 are exempt from the requirements in this subsection.
(5) non-releasable wild animals may be transferred to another individual licensed under this subsection or an individual with a falconry license under 312 IAC 9-10-13 that has had a permit for at least three (3) consecutive years and will use the animal for fostering or educational programs in accordance with this subsection.
(6) A determination that a wild animal is non-releasable must be based upon a licensed veterinarian's examination and statement.
(7) As used in this subsection, "non-releasable" means a wild animal that:
(A) cannot obtain food on its own in the wild,
(B) does not exhibit locomotive skills necessary for that species to survive,
(C) does not possess adequate vision to find/catch food and maneuver in a normal manner, or
(D) cannot otherwise fend for itself without medical care due to disease or extensive injuries.

(p) An individual who does not hold a permit under this section but is listed as an assistant on a permit issued under this section, may assist a permit holder, but only if the permit holder supervises the individual as follows:

(1) An assistant must be at least sixteen (16) years of age to provide assistance at the permit holder's facility.

(2) An assistant under the age of eighteen (18) must be under the direct and on-site supervision of the permit holder.

(3) An assistant of eighteen (18) years of age or older may provide assistance at the permit holder's facility or as follows:

(A) with written authorization an assistant listed on the permit may possess a wild animal under this section at a location not authorized on the permit for no more than thirty (30) consecutive days unless authorized by the department,

(B) the permit holder maintains responsibility for the care of the wild animal,

(C) the permit holder provides supervision with written instructions and is available for phone contact with the assistant at all times,

(D) the facilities of the assistant meet the same facility standards as the permit holder,

(E) the wild animal possessed by an assistant are returned to the permit holder at the time of weaning or release, whichever comes first,

(q) A permit holder may possess a wild animal for rehabilitation under this section for one hundred eighty (180) days except as follows:

(1) Exceptions may be approved by the department but additional time will not be authorized for the sole purpose of releasing the wild animal outside the hunting season for that species.

(2) Migratory birds may only be possessed in accordance with the permit holder's federal migratory bird permit.

(3) Turtles with shell fractures may be possessed for up to two (2) years if housed separately from all other turtles.

(r) All white-tailed deer that are taken in for rehabilitation and determined to be non-releasable must be euthanized. No more than twenty (20) white-tailed deer may be taken in for rehabilitation by a permit holder in one (1) calendar year.

(s) A mammal, non-migratory bird, reptile or amphibian taken in for rehabilitation that is:

(1) diseased; or

(2) severely injured, unless determined by a licensed veterinarian to be treatable and released within one hundred eighty (180) days from the date obtained by the licensed rehabilitator; or

(3) is a turtle with a shell fracture unless determined by a licensed veterinarian to be treatable and released within two (2) years.

must be euthanized.

(t) Euthanasia must be carried out with the:

(1) safest;

(2) quickest; and

(3) most painless

available method as recommended and approved by the division of fish and wildlife.

(u) Carcasses of wild animals that are euthanized or otherwise die while in the care of a permit holder must be:

(1) Buried,

(2) Incinerated on private property with prior permission of the landowner,

(3) Given to a veterinarian or landfill for proper disposal,

(4) Put in the trash if double-bagged,

(5) If euthanized with chemicals, disposed of only according to the chemical label,
(6) Kept for educational purposes only if the permit holder also has a special purpose salvage permit issued under 312 IAC 9-10-13.5,
(7) Donated to a properly licensed university or other educational or scientific institution that has a special purpose salvage permit issued under 312 IAC 9-10-13.5, or
(8) Donated to a person with a permit from the United States Fish and Wildlife Service for Native American religious purposes.

~~(k)~~ **(v)** A permit holder must not:
(1) commercially advertise rehabilitation services or solicit for rehabilitation a wild animal that is subject to this section~~.~~**,**
(2) have a wild animal spayed or neutered or in any way have their reproductive function altered, or
(3) mark or tag the wild animals in any way, such as with a collar or ear-tag, except as required in subsection (n)(2) or in writing from the department.

(w) A permit holder must notify the department within twenty-four (24) hours of acquiring an endangered species or a bald or golden eagle, whether live or dead, and the location and date of acquisition.

(x) A permit holder must notify the department within twenty-four (24) hours if the permit holder has reason to believe that a wild animal has been subjected to criminal activity.

(y) Wild animals, including their carcasses and parts, possessed under this section must not be sold, bartered, or transferred to another individual or to a permit holder's game breeder license except:
(1) as authorized in subsection (u), or
(2) a live wild animal is transferred to another individual with a permit under this section.

(z) A copy of the permit issued under this section must be possessed by the permit holder when transporting a wild animal possessed under this section to another location and be available and on-site when possessing and caring for wild animals authorized under this section.

~~(l)~~ **(aa)** A permit may be suspended, denied, or revoked under IC 4-21.5 if the permit holder fails to comply with any of the following:
(1) A permit issued under this section.
(2) This article or IC 14-22.
~~(3) Another applicable state, local, or federal law.~~
(Natural Resources Commission; 312 IAC 9-10-9; filed May 12, 1997, 10:00 a.m.: 20 IR 2730; readopted filed Jul 28, 2003, 12:00 p.m.: 27 IR 286; filed Sep 23, 2004, 3:00 p.m.: 28 IR 550; readopted filed Nov 24, 2008, 11:08 a.m.: 20081210-IR-312080672RFA)

WILD ANIMAL REHABILITATION PROPOSED RULE CHANGES JUSTIFICATION

Wild animal rehabilitation permits allow qualified individuals to take in wild animals (including mammals, birds, reptiles, and amphibians) for the purpose of caring for the animals until they can be released back into the wild. Many of the animals are orphaned young, but other animals may be sick or injured adults. While wild animal rehabilitators have good intentions, the DNR has had to take legal action on multiple permit holders within the past few years. Violations have included off-site rehabilitation of wild animals by assistants, the failure to release animals, not releasing animals within the timeframe required, filthy and unsanitary rehabilitation facilities that have even warranted action by county health departments, and attempts to transfer of these animals to a game breeder license (which allows the sale of these animals). These issues, as well as others, necessitated the forming of a Wild Animal Rehabilitation Advisory Group to discuss modifying the existing rehabilitation permit rule.

The Wild Animal Rehabilitation Advisory Group that met in 2009 and helped to formulate these proposed changes consisted of the following members: Kathy Hershey (licensed rehabilitator), Kim Hoover (licensed rehabilitator), Ron Johnson (DNR Conservation Officer), Tim Julien (Nuisance Wildlife Control Operator's Association), Dr. Larry McAfee DVM (licensed veterinarian and licensed rehabilitator), Sandra Norman DVM (Indiana Board of Animal Health), Patti Reynolds (licensed rehabilitator), Dean Zimmerman (DNR wildlife biologist), and Linnea Petercheff (DNR, Division of Fish and Wildlife Operations Staff Specialist). The DNR also took input from other rehabilitators who attended the meetings, but were not part of the advisory group. A letter was mailed to all licensed rehabilitators in November of 2009 asking for their input, and many changes were made as a result of their comments.

DNR STATUTORY AUTHORITY

The statutory authority for the DNR Division of Fish and Wildlife in IC 14-22-2-3 is to "(1) Provide for the protection, reproduction, care, management, survival, and regulation of wild animal populations regardless of whether the wild animals are present on public or private property in Indiana; and (2) Organize and pursue a program of research and management of wild animals that will serve the best interests of the resources and the people of Indiana."
The DNR is also required to develop rules that are based upon (A) The welfare of the wild animal, (B) The relationship of the wild animal to other animals, and (C) The welfare of the people in IC 14-22-2-6. Raccoons and coyotes, in particular, create problems for thousands of Hoosiers each year, in addition to posing a significant disease threat and impacting other species of wildlife that the DNR is required to protect.

The DNR is, therefore, proposing to require that mammals that are taken in for rehabilitation and diseased be euthanized for the reasons stated below. Additionally, the DNR is recommending that severely injured mammals be euthanized unless determined by a licensed veterinarian to be treatable and released within 180 days from the date in which the animal was obtained by the licensed rehabilitator.

RACCOON REHABILITATION

1. Diseases and Parasites

Raccoons carry a number of infectious diseases and parasites, some of which can be transferred to humans and domestic animals. Canine distemper is probably the most common disease carried by raccoons and is prevalent in Indiana, possibly due to the high population of raccoons. It can be transmitted to other canines, including domestic dogs (that are not vaccinated) and foxes that come into contact with an infected animal's feces, urine or other bodily secretions, as well as through direct contact with an infected animal. In Missouri in 2009, tests of diseased raccoons showed that approximately 60% had canine distemper. Another study has shown that there is an increase in the prevalence of canine distemper when the raccoons were more closely related, probably due to the fact that direct contact between animals is greater within related animals (Dharmarajan et al. 2010). With a high population of raccoons and the fact that they will den together in trees and other enclosed spaces in cold weather, the potential for disease transmission is high. Some raccoons taken in by rehabilitators have also been found to have raccoon parvovirus. The DNR has received multiple reports from licensed rehabilitators of raccoons dying from this disease while in their possession. No diseased raccoons should be rehabilitated and released back into the wild. While many rehabilitators treat these animals with vaccinations for canine distemper, parvovirus, and rabies, none of these vaccines are labeled for use on wild animals and have not been proven to be effective. This treatment usually does not even save the animal. By keeping these diseased animals in captivity, the disease can be spread to other animals within the same facility. Many rehabilitators also house raccoons together that come from different areas, spreading the disease to other raccoons in their possession. New information has revealed that some rehabilitators in Indiana are using Tamiflu©, an antiviral drug labeled for influenza in humans, on raccoons with distemper. Public health professionals have recommended that these drugs NOT be used in wildlife, and the FDA prohibits the use of this drug for extralabel use in chickens, turkeys and ducks. Additionally, the American Veterinary Medical Association stated in 2008 that no drug is available that will kill the canine distemper virus in infected dogs. If the raccoons are not housed properly to prevent the spread of disease, releasing raccoons with parvovirus or distemper provides for the transmission of these diseases to wild populations and may be to areas where the disease is not currently found. Attached please find one report from a licensed rehabilitator that documents a parvovirus outbreak.

Raccoons can also carry the roundworm, *Baylisascaris procyonis*, which can be transferred to humans and domestic animals through the ingestion of infected eggs. This roundworm, if ingested through soil, water, or on objects that have been contaminated with raccoon feces, can be fatal to humans, especially small children that will put almost anything they find into their mouths (such as raccoon feces found in sandboxes). Other animals can also become infected (such as dogs) by eating a small animal that has been infected with this roundworm (Sorvillo et al. 2002). Raccoons are also known to frequent barn lofts and defecate on top of stored straw or hay bales. As these bales are tossed about, the potential for raccoon roundworm eggs to become airborne and inhaled in greatly increased. Research from a professor at Purdue University has documented this parasite in Indiana raccoons. Furthermore, ongoing research in several counties in northern Indiana has found that a large proportion of raccoon scat carry this roundworm. Allegheny woodrats, an endangered species in Indiana, experience mortality as a result of infection by raccoon roundworm, in addition to other factors. Attached also please find information about this roundworm and its devastating affects.

Raccoons can also carry rabies, of which there are different strains. The Centers for Disease Control and Prevention (see attached articles) recommended in 2007 that "stronger and more uniform federal and state wildlife regulations are necessary to prevent indiscriminate international, interstate, and intrastate movement and release of wild carnivores by private citizens." The CDC also reports, "Translocation of infected animals, as has occurred in the past, continues to pose a substantial threat to the goals of national oral rabies vaccination programs." The CDC has reported that the numbers of raccoons with rabies has decreased since 2004. However, the CDC reports, "enzootic transmission of rabies among raccoons and from rabid raccoons to other species continued in 20 states, New York City, and the District of Columbia in 2008." Although the raccoon rabies strain is not yet in Indiana, it is in Ohio and has spread to the Cleveland area within recent years. Oral rabies vaccinations, which are costly, have been used in states such as Ohio to prevent the spread of this raccoon rabies strain. Raccoons can also carry leptospirosis and other parasites.

"Movement of both rabid and non-rabid raccoons plays in important role in disease spread." (Rosatte et. al., 2010)

Research was done in 1993 and 1994 in Illinois regarding the movements of translocated raccoons. "Because most of the translocated raccoons did not stay at the release site and sometimes dispersed considerable distances, translocated animals could add to nuisance wildlife problems for rural human residents or increase the spread of disease during zoonotic outbreaks. Hence, because some translocated animals disperse for long distance after translocated, the rate of spread of zoonotic disease could be accelerated considerably. Further research on the effects of translocated raccoons is warranted because of the potential consequences for other wildlife species." (Mosillo et. al. 1999)

Another study found that where the population of raccoons was manipulated to be higher, the prevalence of raccoon roundworm increased to 54%, significantly greater than prevalence in the same subpopulation before the raccoons had greater intraspecific contact (Gompper et. al. 2005).

While some rehabilitators treat the raccoons in their care with vaccines for canine distemper, parvovirus, and rabies, none of these vaccines have been proven or documented to be effective for the treatment of these diseases in raccoons or other wild animals and are not labeled for use on wild animals.

2. <u>Predation on other species</u>
The raccoon population in Indiana is thought to be very high, which can have a substantial impact on other species of wildlife, such as ground-nesting birds and turtles. Some ground-nesting birds, such as bobwhite quail, can be significantly impacted by predators such as raccoons and coyotes. When a predator approaches, wildlife (such as ground-nesting birds)

tend to move away from their eggs to try to lure the predator away from them, but it often results in the destruction of the eggs. Some of these species already have limited habitat in Indiana.

Various studies on predation of game birds have been conducted in the United States, with differing results, depending on the habitat and availability of other prey species. However, in one study, predation was the primary cause of mortality for bobwhites at all life stages (Rollins and Carroll 2001).

Raccoons are also common predators of turtle nests. Most species of turtles in Indiana bury their eggs in a hole in the ground in moist soil or sand near water. While some young hatch in the year the eggs are laid, others over-winter in the nest, making them very vulnerable to predation. One study done in Michigan showed that predation accounted for 82% of turtle nest mortality (Congdon et al. 2009) and another study in Iowa reported that over 80% of natural nests were destroyed by predators (Nelson et al. 2009). The most common predators of turtle eggs are raccoons, skunks, foxes, and opossums. Egg-laying snakes also lay their eggs in sand or soil where they are also subject to predation. Six (6) species of turtles and three (3) species of egg-laying snakes are classified as endangered in Indiana with 3 additional species listed as special concern.

Raccoons are also common on farms where they destroy corn, get into barns, kill chickens, and cause other damage. The DNR has already received a letter from a representative of the Farm Bureau about the problems that raccoons are causing to farmers.

Additional evidence of a high raccoon population in Indiana includes the numbers of nuisance raccoons taken by nuisance wild animal control permit holders, number of raccoons sold to licensed fur buyers, numbers of calls to the wildlife conflicts hotline in previous years, the DNR raccoon roadkill survey index, and amount of distemper outbreaks among raccoons throughout Indiana.

3. Nuisance Animals

Raccoons are also considered to be a nuisance animal by many landowners in urban and suburban neighborhoods. Raccoons will enter attics, barns, sheds, and fireplaces to live and have their young. They will destroy soffitts in an effort to gain access to attics. Licensed nuisance wild animal control operators dealt with 7,275 raccoons in 2008 (5,074 were killed/euthanized) and 4,484 raccoons (3,031 were killed/euthanized) in 2007. USDA – Wildlife Services staff, manning the Wildlife Conflicts Information Hotline, handled approximately 1,000 raccoon-related calls each year for the past few years. Licensed rehabilitators released 890 raccoons in 2008 and 852 in 2007. Licensed rehabilitators lost another 582 raccoons due to disease or were euthanized in 2008 and another 285 died or were euthanized in 2007.

N. Budd Veverka reported in the Indiana 2008 & 2009 Raccoon Road-kill Survey Report (2010), "The 2008 and 2009 statewide raccoon road-kill indices, though not the highest observed, continue a steady upward trend, likely representing an increasing raccoon population. Regionally, data indicates an increasing trend in the relative abundance of raccoons for all six regions across the state, with record highs in the Southwest and South Central regions in 2009. Due to the collapse of the fur market in the late 1980s and the cultural decline in trapping, the raccoon population in Indiana has experienced significant growth over the past two decades."

"Urban raccoons experienced the fewest mortality sources, whereas rural raccoons experienced the most. Disease was the greatest mortality factor at the urban site, while vehicle-related mortalities dominated at the suburban and rural sites. Our data suggest that multiple factors, including increased survival, higher annual recruitment, and increased site fidelity, are jointly responsible for high-density raccoon populations in urbanized areas." (Prange et al. 2003)

"In suburban areas, raccoons often raid gardens and garbage cans, and carrion is readily consumed. They will also feed on small vertebrates, such as young turtles or muskrats, when these are readily available, and dig up the eggs of .. turtles in large numbers on some nesting beaches." (Whitaker and Hamilton 1998)

4. Legal Impacts

Some of Indiana's state legislators have also expressed concerns about the numbers of raccoons and complaints they have received regarding raccoon damage, and the Indiana Farm Bureau has also sent a letter to the DNR regarding crop damage from raccoons.

5. Conclusion

The majority of raccoons taken in by rehabilitators are orphaned young. Raccoons can have 3-7 young each year, and typically live up to 5 years in the wild. Foxes, bobcats, coyotes, owls, and other predators kill some juvenile raccoons, but for adult raccoons, automobiles, diseases and accidents (such as from falling from trees) are more important causes of death than are predators (Whitaker and Hamilton 1998).

Limiting the numbers of raccoons released by wildlife rehabilitators and requiring raccoons that are diseased (distemper, parvovirus, etc.) to be euthanized shows that the DNR is taking action to provide for the welfare of wild animal populations, including that of raccoons, coyotes and other species (turtles, ground-nesting birds, etc.). This step helps provide for the protection, reproduction, management, and survival of many other species of wild animals that the DNR has the responsibility of protecting, without impacting the raccoon population to such a level that they become rare. This step also helps provide for the welfare of the citizens of Indiana by helping to limit the spread of disease. The DNR also has the authority in IC 14-22-2-6 to promulgate administrative rules that provide for: (A) The welfare of the wild animal; (B) The relationship of the wild animal to other animals; (C) The welfare of the people.

REHABILITATION OF OTHER MAMMALS

1. Disease and parasites

Virginia opossums, foxes, striped skunks, and coyotes also carry a number of infectious diseases and parasites, some of which can be transferred to humans and domestic animals.

Coyotes and red foxes can carry canine distemper, the tapeworm *Echinococcus multilocularis* (a tapeworm which also has public health implications), and canine heartworm; distemper is especially noticeable when populations are high. Canine distemper can be transmitted to other canines, including domestic dogs. Striped skunks, foxes, opossums, and coyotes can also carry rabies, of which there are different strains. Coyotes can also carry parvovirus enteritis and brucellosis, while Virginia opossums can also carry Sarcocystis neurona, a protozoan parasite that causes equine protozoal myeloencephalitis (EPM), which can be fatal to horses. EPM is costly to treat, and not all horses recover from it. Beavers can also carry tularemia and parasites that carry *Giardia.* Rehabilitators often treat coyotes and other wild animals in their care with vaccines for canine distemper, parvovirus enteritis, and rabies, none of these vaccines have been proven or documented to be effective for the treatment of these diseases in wild animals and are not labeled for use on wild animals.

2. <u>Predation on other native species</u>

Virginia opossums, foxes, striped skunks, and coyotes impact other native species in Indiana. The coyote population in Indiana is doing well and reports of coyotes in urban and suburban areas appear to be increasing. Coyotes have also been known to kill dogs, cats, and livestock. Virginia opossums, foxes, striped skunks, and coyotes will also kill poultry and eat their eggs.

Some ground-nesting birds, such as bobwhite quail, can be significantly impacted by predators such as raccoons and coyotes. Various studies on predation of game birds have been conducted in the United States, with differing results, depending on the habitat and availability of other prey species. However, in one study, predation was the primary cause of mortality for bobwhites at all life stages (Rollins and Carroll 2001). Ground-nesting birds are especially vulnerable to predation, and the most common predators include skunks, raccoons, opossums, bobcats, foxes, and coyotes (Hernandez et al. 1997, Fies and Puckett 2000, Staller et al. 2005). Stanley D. Stewart, a wildlife biologist in Alabama, stated, "If habitat is good, predator management can carry quail population to higher densities." When a predator approaches, wildlife tend to move away from their eggs to try to lure the predator away from the nest, but it often results in the destruction of their eggs.

The most common predators of turtle eggs are raccoons, skunks, foxes, and opossums. Most species of turtles in Indiana bury their eggs in a hole in the ground in moist soil or sand near water. While some young hatch in the year the eggs are laid, others over-winter in the nest, making them very vulnerable to predation. One study done in Michigan showed that predation accounted for 82% of turtle nest mortality (Congdon et al. 2009) and another study in Iowa reported that over 80% of natural nests were destroyed by predators (Nelson et al. 2009). Egg-laying snakes also lay their eggs in sand or soil where they are also subject to predation. Six (6) species of turtles and three (3) species of egg-laying snakes are classified as endangered in Indiana with 3 additional species listed as special concern.

3. <u>Nuisance Animals</u>

Coyotes, red foxes, opossums, and striped skunks are also considered to be a nuisance animal by many landowners in urban and suburban neighborhoods. Coyotes, in particular, are seen more often in suburban neighborhoods and many people are afraid for their children and pets. Years ago, the Indiana General Assembly passed a law (IC 14-22-6-12) to allow landowners, and anyone authorized in writing by a landowner, to take coyotes on their land at any time to help deal with nuisance coyotes.

LOCATIONS WHERE WILD ANIMALS CAN BE RELEASED

The Wild Animal Rehabilitation Advisory Group and DNR believe that restrictions need to be in place for all species in regard to where they can be released. However, the greatest need for location restrictions concern the release of raccoons, foxes, coyotes, skunks, and opossums for the following reasons:

1. All of these species carry a variety of diseases that can be transmitted to humans and/or domestic animals. Raccoons, foxes, and coyotes can carry canine distemper, parvovirus, rabies, and leptospirosis. Virginia opossums can also carry Sarcocystis neurona, a protozoan parasite that causes equine protozoal myeloencephalitis (EPM), which can be fatal to horses. EPM is costly to treat, and not all horses recover from it. Opossums are believed to be the main carrier of EPM, and horse owners are advised to keep barns, pastures, and stable areas free of opossums to help lower the risk of EPM (Midwest Thoroughbred 2010). Striped skunks also carry canine distemper, the ascarid roundworm, and leptospirosis. Since there are no vaccines proven or labeled to be effective for treating rabies, distemper or parvovirus in wild animals, there is no guarantee that the release of these species of animals will not transfer a disease or parasite to other wild populations since they may have been held in enclosures with or near other diseased animals from other counties.
2. All of these species are found throughout Indiana. Therefore, releasing these animals in areas where other animals of the same species already exist increases competition for food, water, and shelter for the animals already present, or will cause those animals to disperse and have to cross roads where their likelihood of survival is minimal. Wildlife habitat has a carrying capacity and releasing large numbers of animals of the same species on the same property where other animals of the same species already live only increases competition for food, water, and shelter.

Resident landowners and tenants who take a wild animal that is causing damage on their property can kill the animal or release it, but if released, it must be released in the same county (312 IAC 9-3-15). Nuisance wild animal control permit holders already must release raccoons and <u>all</u> other wild animals that they capture in the same county of capture or euthanize them (312 IAC 9-10-11).

In an article on the rehabilitation of raccoons (White 1989), "The numbers of raccoons released in a given area should be tightly controlled so that their impact on the area is minimized."

RELEASE WITHIN 180 DAYS

The new language governing the requirement to release a wild animal taken in for rehabilitation within 180 days is currently in a non-rule policy that was approved by the Natural Resources Commission in May of 2006 (see attached). Currently, wild animals must be released or euthanized within 180 days of taking possession of the animal, unless an exemption is granted by the DNR (conservation officer or Operations Staff Specialist). The time of year is not an approvable reason for an extension, which has been requested in the past by rehabilitators for several species, especially white-tailed deer.

Migratory birds must be released within 180 days according to federal law (50 CFR 21.31), unless additional time is authorized by the U.S. Fish and Wildlife Service Migratory Bird Permit Office. Other states such as Kansas and Wisconsin already have limits on the number of days a wild animal can be possessed by a licensed rehabilitator.

Keeping animals longer than this date only provides more opportunities for the animals to be become habituated to humans, pick up more diseases or parasites, and become treated like pets. Wild animals were not designed for life in a cage and should not be help captive longer than necessary.

CONCLUSION

In conclusion, the majority of the proposed changes in this rule were made by an advisory group that included a diverse membership, including a trapper (nuisance wild animal control operator), conservation officer, wildlife biologist, veterinarian, and wild animal rehabilitators. The changes were agreed upon by consensus of the group. Trained, certified wildlife biologists believe that these changes will help provide for healthier populations and reduce predation on species that are already in peril. These changes are needed to ensure that wild animals are being housed and cared for properly, and are not affecting the welfare of wild animal populations.

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Fact Sheet for the general public

Baylisascaris Infection
(bay-liss-ass-kuh-ris)
Raccoon Roundworm Infection

Division of Parasitic Diseases

What is *Baylisascaris* infection?

Baylisascaris, an intestinal raccoon roundworm, can infect a variety of other animals, including humans. The worms develop to maturity in the raccoon intestine, where they produce millions of eggs that are passed in the feces. Released eggs take 2-4 weeks to become infective to other animals and humans. The eggs are resistant to most environmental conditions and with adequate moisture, can survive for years.

How do humans become infected?

People become infected when they accidentally ingest infective eggs in soil, water, or on objects that have been contaminated with raccoon feces.

When humans ingest these eggs, they hatch into larvae in the person's intestine and travel throughout the body, affecting the organs and muscles.

Who is at risk for infection?

Anyone who is exposed to environments where raccoons live is potentially at risk. Young children or developmentally disabled persons are at highest risk for infection when they spend time outdoors and may put contaminated fingers, soil, or objects into their mouths. Hunters, trappers, taxidermists, and wildlife handlers may also be at increased risk if they have contact with raccoons or raccoon habitats.

How common is *Baylisascaris* infection in raccoons?

Fairly common. Infected raccoons have been found throughout the United States, mainly in the Midwest, Northeast, middle Atlantic, and West coast. Infection rarely causes symptoms in raccoons. Predator animals, including dogs, may also become infected by eating a smaller animal that has been infected with *Baylisascaris.*

How do raccoons become infected?

Raccoons become infected in one of two ways:

- Young raccoons become infected by eating eggs during foraging, feeding, and grooming.
- Adult raccoons acquire the infection by eating rodents, rabbits, and birds infected with the larvae of *Baylisascaris.*

How common is *Baylisascaris* infection in humans?

Infection is rarely diagnosed. Fever than 25 cases have been diagnosed and reported in the United States as of 2003. However, it is believed that cases are mistakenly diagnosed as other infections or go undiagnosed. Cases have been reported in Oregon, California, Minnesota, Illinois, Michigan, New York, and Pennsylvania. Five of the infected persons died.

What are the symptoms of *Baylisascaris* infection in humans?

Symptoms of infection depend on how many eggs are ingested and where in the body the larvae migrate (travel to). Once inside the body, eggs hatch into larvae and cause disease when they travel through the liver, brain, spinal cord, or other organs. Ingesting a few eggs may cause few or no symptoms, while ingesting large numbers of eggs may lead to serious symptoms. Symptoms of infection may take a week or so to develop.

Symptoms include

- Nausea
- Tiredness
- Liver enlargement
- Loss of coordination
- Lack of attention to people and surroundings
- Loss of muscle control
- Coma
- Blindness

Other animals (except raccoons) infected with *Baylisascaris* can develop similar symptoms, or may die as a result of infection.

What should I do if I think I have ingested *Baylisascaris* eggs?

If you suspect you have been infected, consult your health care provider immediately. Be sure to report that you have recently been exposed to raccoons or their feces.

How is infection diagnosed?

Infection is difficult to diagnose and often is made by ruling out other infections that cause similar symptoms. Information on diagnosis and testing can be obtained through DPDx or your local health department.

How should I clean up raccoon feces?

You should clean up very carefully. To eliminate eggs, feces and material contaminated with raccoon feces should be removed and burned, buried, or sent to a landfill. Care should be taken to avoid contaminating hands and clothes. The use of gloves and facemask will help prevent cross contamination. Treat feces-soiled decks, patios, and other surfaces with boiling water. Always wash hands well with soap and running water, to help further reduce possible infection.

Is treatment available?

Early treatment might reduce serious damage caused by the infection. Should you suspect you may have ingested raccoon feces, seek immediate medical attention.

How can I prevent infection in myself, my children, or my neighbors?

- Avoid direct contact with raccoons -- especially their feces. Do not keep, feed, or adopt raccoons as pets! Raccoons are wild animals.
- Discourage raccoons from living in and around your home or parks by
  - preventing access to food
  - closing off access to attics and basements
  - keeping sand boxes covered at all times, (becomes a latrine)
  - removing fish ponds – they eat the fish and drink the water

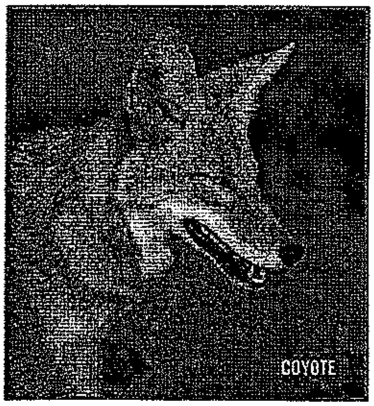
  - eliminating all water sources
  - removing bird feeders
  - keeping trash containers tightly closed
  - clearing brush so raccoons are not likely to make a den on your property.
- Stay away from areas and materials that might be contaminated by raccoon feces. Raccoons typically defecate at the base of or in raised forks of trees, or on raised horizontal surfaces such as fallen logs, stumps, or large rocks. Raccoon feces also can be found on woodpiles, decks, rooftops, and in attics, garages, and haylofts. Feces usually are dark and tubular, have a pungent odor (usually worse than dog or cat feces), and often contain undigested seeds or other food items.
- **To eliminate eggs, raccoon feces and material contaminated with raccoon feces should be removed carefully and burned, buried, or sent to a landfill.** Care should be taken to avoid contaminating hands and clothes. Treat decks, patios, and other surfaces with boiling water or a propane flame-gun. (Exercise proper precautions!) Newly deposited eggs take at least 2-4 weeks to become infective. Prompt removal and destruction of raccoon feces will reduce risk for exposure and possible infection.
- Contact your local animal control office for further assistance.

This fact sheet is for information only and is not meant to be used for self-diagnosis or as a substitute for consultation with a health care provider. If you have any questions about the disease described above or think that you may have a parasitic infection, consult a health care provider.

Revised September 23, 2004

What is Canine Distemper?

Canine distemper is a highly contagious and serious disease caused by a virus that attacks the respiratory, gastrointestinal, and, often, the nervous systems of puppies and dogs. The virus also infects wild canids (e.g. foxes, wolves, coyotes), raccoons, skunks, and ferrets.



How is Canine Distemper virus spread?

Puppies and dogs usually become infected through airborne exposure to the virus contained in respiratory secretions of an infected dog or wild animal. Outbreaks of distemper tend to be sporadic. Because canine distemper also affects wildlife populations, contact between wild canids and domestic dogs may facilitate spread of the virus.

What dogs are at risk?

All dogs are at risk but puppies younger than four months old and dogs that have not been vaccinated against canine distemper are at increased risk of acquiring the disease.

What are some signs of Canine Distemper?

The first sign of distemper is eye discharge that may appear watery to pus-like. Subsequently, dogs develop fever, nasal discharge, coughing, lethargy, reduced appetite, vomiting, and diarrhea. In later stages, the virus may attack the nervous system, bringing about seizures, twitching, or partial or complete paralysis. Occasionally, the virus may cause footpads to harden. Distemper is often fatal. Even if a dog does not die from the disease, canine distemper virus can cause irreparable damage to a dog's nervous system. Distemper is so serious and the signs so varied that any sick dog should be taken to a veterinarian for an examination and diagnosis.

How is Canine Distemper diagnosed and treated?

Veterinarians diagnose canine distemper on the basis of clinical appearance and laboratory tests. No specific drug is available that will kill the virus in infected dogs. Treatment consists primarily of efforts to prevent secondary infections; control vomiting, diarrhea, or neurologic symptoms; and combat dehydration through administration of fluids. Ill dogs should be kept warm, receive good nursing care, and be separated from other dogs.

How is Canine Distemper prevented?

Vaccination and avoiding contact with infected animals are key elements of canine distemper prevention.

Vaccination is important. Young puppies are very susceptible to infection, particularly because the natural immunity provided in their mothers' milk may wear off before the puppies' own immune systems are mature enough to fight off infection. If a puppy is exposed to canine distemper virus during this gap in protection, it may become ill. An additional concern is that immunity provided by a mother's milk may interfere with an effective response to vaccination. This means even vaccinated puppies may occasionally succumb to distemper. To narrow gaps in protection and optimally defend against canine distemper during the first few months of life, a series of vaccinations is administered.

Until a puppy has received its complete series of vaccinations, pet owners should use caution when taking their pet to places where young puppies congregate (e.g. pet shops, parks, puppy classes, obedience classes, doggy daycare, and grooming establishments). Reputable establishments and training programs reduce exposure risk by requiring vaccinations, ealth examinations, good hygiene, and isolation of ill puppies and dogs.

To protect their adult dogs, pet owners should be sure that their dog's distemper vaccination is up-to-date. Ask your veterinarian about a recommended vaccination program for your canine companion.

Contact with known infected dogs should always be avoided. Similarly, contact with raccoons, foxes, skunks, and other potentially infected wildlife should be discouraged.

Although this brochure provides basic information about canine distemper, your veterinarian is always your best source of health information. Consult your veterinarian for more information about canine distemper and its prevention.

And Now A Note On Your Pet's *General Good Health*

A healthy pet is a happy companion. Your pet's daily well-being requires regular care and close attention to any hint of ill health. The American Veterinary Medical Association suggests that you consult your veterinarian if your pet shows any of the following signs:

- Lumps or swelling
- Reduced or excessive appetite or water intake
- Marked weight loss or gain
- Limping, stiffness, or difficulty getting up or down
- Difficult, discolored, excessive or uncontrolled waste elimination (urine and feces)
- Abnormal discharges from any body opening
- Head shaking, scratching, licking, or coat irregularities
- Changes in behavior or fatigue
- Foul breath or excessive tartar deposits on teeth

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Recommendations of a national working group on prevention and control of rabies in the United States

Article III: Rabies in wildlife

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The complexity of controlling rabies has increased dramatically in the United States since wildlife began to replace domestic dogs as the principal disease vector > 40 years ago.[1] Extensive use of veterinary clinics for parenteral vaccination of domestic dogs, observation of the suspect biting animal, and public education campaigns (together with the application of **postexposure prophylaxis [PEP]** following a dog bite) during the 1950s and 1960s were effective, simple strategies for the management of rabies in dogs. However, these strategies were not directly applicable to the management of rabies in wildlife.

Management of rabies in wildlife is complicated by the ecologic and biologic factors associated with wildlife reservoirs, the multiagency approach needed to manage an important public health problem originating in wildlife, the limitations of available control methods, and the broad range of public attitudes toward wildlife. In addition, there are several variants of the rabies virus in the United States.[2,3] These are associated with terrestrial carnivores, including raccoons, skunks, and arctic, red, and gray foxes; a number of variants are also found in a variety of species of bats. Recently, an apparent viral shift or adaptation developed with a variant of the virus in canids at the United States-Mexico border area, resulting in local transmission among coyotes and dogs in south Texas.[4] Compounding the problem, animals infected with this variant were translocated to other states.[5] There are equally important biologic, behavioral, and ecologic differences among carnivores that imply inherent differences in disease control approaches to each major reservoir.

It is often difficult to examine the precise epizootic characteristics of rabies as it spreads among animals of a given population. Also, the role of reservoir host abundance and demography is poorly understood. However, it is possible to make several generalizations from passive surveillance data and trends in the reporting of cases of rabies in animals, especially from detailed studies of red fox populations in Europe. Where measures of carnivore abundance exist, the incidence of rabies in animals (presumably all species) is often positively associated with the increasing density of a dominant reservoir species.[6] In general, rabies epizootics affecting carnivores spread in wavelike fashion; the area experiencing the current epizootic is the crest, and the locale with low reservoir-host populations is the trough.[7] Rabies is viewed as a density-dependent disease, and population dynamics of reservoir hosts are regarded as critical to understanding and modeling the temporal and spatial patterns of rabies in wildlife.[8,9] In part, the rate of spread of rabies in populations of a particular species can be related to, or modeled by, its social structure, dispersal patterns, and contact rates.[8,10,11] Habitat features, such as continuity and patch size, may play a role in the rate of spread and persistence of the disease.[12,13] Furthermore, interactions among species complicates the understanding of the ecologic and epidemiologic factors associated with rabies.[14] Spillover of infection from the dominant reservoir of a region to other species has been documented,[2] but the processes by which new variants and epizootics of rabies virus emerge in different reservoir species are unknown. After an epizootic has abated, terrestrial reservoir populations decrease, and reports of rabies in animals in a given locale can decline precipitously. An apparent host threshold density has been suggested as necessary for rabies to perpetuate in red fox populations.[15] Below this threshold, contacts appear too few to continue transmission. The threshold phenomenon has not been well studied in other species, although it is presumed to exist. In addition, immunity in surviving members of the reservoir population has been suggested for several species following an epizootic of rabies.[14] The influence of herd immunity and population recovery time in the recrudescence of rabies in enzootic areas are largely unstudied for the terrestrial rabies reservoirs; the situation is similar for the various species of bats and diverse rabies virus variants of bats.

There are a number of approaches for management of rabies in wildlife, such as reduction of vector populations, modification of habitats, parenteral vaccination through **trap-vaccinate-release (TVR)** programs, oral vaccination, and passive disease surveillance. In addition, immunocontraception for limiting wildlife populations and modulating density-related disease is an intriguing and rapidly progressing area of research, although it is still considered highly experimental.[16] These various methods could be used alone or in combination. The utility of wildlife control methods, either independently or in an integrated control program, will depend in large part on the overall objectives. Objectives for the control of rabies may vary regionally, from state to state, and within a state. State wildlife agencies with the statutory authority to manage wild animals may not universally endorse the same management strategy as their respective state public health agencies. Moreover, the existence of rabies reservoirs in multiple states (with the exception of mongooses and their unique rabies virus variant in Puerto Rico) makes strategic control of rabies a national issue. Oral vaccination programs for carnivores are in developmental or early operational stages, and their field efficacy, costs, and benefits remain uncertain. Conventional management approaches, such as long-term, federally supported population reduction on a broad scale, no longer appear justified relative to social acceptability, cost, and efficacy. Lastly, wildlife reservoir species, unlike domestic animals, are natural resources; therefore, public input is essential in helping to determine the methods used to manage rabies among these animals.[17]

Rabies Vector Population Reduction

Historically, population reduction has been used to control rabies on the premise that densities of susceptible animals can be reduced below a threshold necessary for rabies to spread through populations.[18] The efforts to control rabies in skunks in Alberta, Canada represents one of the only recent, documented, and broader-scale uses of population reduction in North America conducted explicitly to control rabies. Success of the control program in Alberta was attributed to a high level of effort during several years, the well-defined behavior of skunks in prairie habitats, and access to an effective method.[19] Compensatory changes in carnivore reproduction and dispersal can limit the effectiveness of controlling population numbers of other species in different conditions.[9,20]

Population reduction with toxicants is impractical as a broadscale control alternative for rabies in the United States. Presidential Executive Order 11643, issued in 1972, banned the use of toxicants (compound 1080, strychnine, sodium cyanide, and thallium sulfate) for control of predators.[21] The M-44 cyanide capsule has been reregistered with some applicability for controlling rabies vectors (coyotes and red and gray foxes).[22] In addition, research conducted by USDA Animal Damage Control (recently renamed Wildlife Services) has led to the development of a large gas cartridge that may be used for lethal elimination in dens of skunks, coyotes, and red foxes. Similarly, there is a commercially produced gas cartridge for use on denning skunks (also with moles, woodchucks, or other rodents). Various types of traps and aerial and ground shooting could potentially be used with toxicants in an integrated population reduction strategy to control rabies in some species. However, trapping and shooting options for population reduction of wildlife species would require the opportunity for extensive review by and input from all potentially affected stakeholders.

The estimated costs of population reduction vary widely[18,23]; however, such efforts would most likely be cost prohibitive if programs relied on labor-intensive trapping and shooting.[18] Other issues, such as impacts to nontarget species[24] and limited public support for population reduction efforts, clearly restrict the feasibility of this approach as a single tactic for broad-scale control of rabies. Presently, population reduction is most likely to be publicly accepted and effective in localized or site-specific scenarios in the United States (eg, reducing the density of raccoon populations in parks where visitors may come in contact with potentially rabid animals). The use of intensive local population reduction as a contingency to address outbreak foci remains untested. Population reduction also may continue to merit consideration for species or situations in which all other methods are not practical.

Habitat Modification

Habitat modification is a useful site-specific management approach that can reduce the chance of interaction between human beings and potential vectors like skunks, raccoons, and bats. Managing refuse through routine garbage pickup, using animal-proof garbage receptacles, making pet food inaccessible to wild animals, capping chimneys, and screening louver vents are examples of habitat manipulation to minimize contact between wild animals, pets, and people. Techniques designed to prevent access of bats to human living quarters can minimize PEP considerations.

Trap-Vaccinate-Release Programs

A TVR program was implemented in Toronto in 1984[11] as an interim measure to control rabies in skunks until an **oral rabies vaccine (ORV)** could be developed. Inactivated rabies virus vaccine was injected IM into live-trapped skunks. Serum samples from recaptured animals indicated that a high level of population immunity was effective in reducing rabies in Toronto. Costs were relatively high ($450 to $1,150/sq km [US dollars]) for the TVR program in Toronto, but these costs may be offset partially by a reduction in the number of people receiving PEP.[25] Similarly, there have been TVR programs targeting raccoons on the Delmarva (Delaware, Virginia, and Maryland) peninsula,[a] in Philadelphia,[b] and in Ithaca, NY.[c] A TVR program is also being implemented along the Ontario boundary of the Niagara Frontier in an attempt to provide a barrier against the invasion of rabid raccoons.

Oral Rabies Vaccination

After the concept was conceived at the **Centers for Disease Control and Prevention (CDC)** during the 1960s, oral vaccination of red foxes with attenuated Evelyn-Rokitnicke-Abelseth vaccine was demonstrated in the early 1970s.[26] The intensity of rabies in foxes in Europe stimulated the further development of vaccines and delivery systems,[27] and these efforts were supported by the World Health Organization. Early control efforts included ground and aerial delivery of rabies vaccine in blister packs inserted in chicken head baits. Oral vaccination in Europe has since progressed to include the use of several efficacious vaccines, including attenuated and recombinant rabies vaccines, in a variety of commercial baits; these are distributed through a combination of ground and aerial bait distribution methods.[28] During 1999, Switzerland, which had a long enzootic for rabies in red foxes and was the first country to use ORV in wildlife in 1978, was declared free of rabies.

In 1989, a similar ORV program was implemented in southern Ontario. Efforts in Ontario throughout the past decade have been promising and have greatly advanced aerial bait delivery with fixed-wing aircraft over large, homogenous areas of land.[29] The ultimate objective of eliminating the arctic fox variant of the rabies virus, which is transmitted among red foxes in the region, may hinge in part on cooperation with neighboring northeastern states and provinces.

Although oral vaccination shows promise for control of rabies among some terrestrial vectors, many important questions regarding baits, optimal baiting strategies, and relevant techniques for control of rabies in bats remain unanswered.[1] Subsequent to placebo baiting studies to evaluate the feasibility of oral vaccination,[30-32] the potential benefits of oral vaccination have been questioned.[33] In contrast, the public is often supportive of novel control methods,[34] despite the infancy of oral vaccination for control of rabies among terrestrial vectors in the United States. To warrant consideration as a public health intervention, efficacy of oral vaccination must be proven, and desirable cost-benefit ratios or a willingness to pay among the general public or other stakeholders will be required.[35-36]

In the United States, international and multidisciplinary collaborative efforts led to the development of a **vaccinia-rabies glycoprotein (V-RG)** recombinant virus vaccine for use in raccoons.[37-42] In 1997, the results of safety and efficacy field trials led to full licensure of the V-RG vaccine for use in state or federally approved oral vaccination projects targeting raccoons.[43] To date, the V-RG vaccine has been used in > 10 completed or ongoing field projects for control of rabies in raccoons.[1,44] The potential effectiveness of oral vaccination at containing epizootic fronts was first described in New Jersey[45] at the Cape May peninsula and subsequently at the Cape Cod isthmus in Massachusetts.[46] The first suppression of rabies in an enzootic area was described in the Capital Region of New York.[1] Additional ongoing pilot studies have yet to generate substantial data on which to base operational plans for control of rabies through oral vaccination.[1] However, it is clear that federal involvement in strategic oral vaccination efforts involving multiple states will be essential if the ultimate goal is elimination of a particular terrestrial variant of rabies virus. In addition to pilot studies for control of rabies in raccoons, the V-RG vaccine is also being used in an effort to prevent the spread of rabies among gray foxes in west Texas and among coyotes in south Texas.[47]

Surveillance

Surveillance is integral to all efforts of rabies control. Surveillance should be pursued more aggressively and with an analytic design during control field trials to objectively evaluate effectiveness. National typing of rabies virus variants should be continued, because such efforts would lead to better understanding of the spatial and temporal distribution of different variants. Such information is essential in view of the differences in behavior and population dynamics and structure among the major wildlife vectors. If regional control efforts directed at specific variants of the virus are initiated, historic and current surveillance data on variants will be needed. Basic passive surveillance will be insufficient for monitoring the effect of oral vaccination on rabies in wildlife.

Contraception

Interest in oral contraception in wildlife began in the early 1960s as a means to control coyote populations causing livestock depredations in the west and red fox populations responsible for the spread of rabies in the eastern United States.[48] Research efforts with the reproductive inhibitor diethylstilbestrol had promise,[49] but these were abandoned because of the lack of safe, effective, long-lasting agents, and effective delivery systems. Recently, contraception has regained attention as a means of controlling wildlife populations.[16] Advances in genetic technology since the 1960s have led to the development of immunocontraceptives.[50-52] Nevertheless, field delivery of oral immunocontraceptives presents many of the same challenges that confronted researchers of oral rabies vaccination in the 1960s. Much work remains to develop safe and effective delivery systems.[48,50,53,54] In addition, many stakeholders will have a voice in defining the conditions under which immunocontraceptives could be acceptably applied.[55]

Recommendations—A better understanding of the complex interaction of host factors (eg, density dependent changes in reproduction, survival, and dispersal, and level of immunity in the surviving population) and viral characteristics involved in epizootic and enzootic transmission of rabies in wildlife populations is necessary. Surveillance systems that include detailed ecologic or epidemiologic data are needed. Explicit descriptions of the impact of rabies on the population dynamics of carnivores and the potential effect of interventions, such as oral vaccination, are fundamentally lacking and critically needed. Educational materials for the public on rabies in wildlife and potential control measures also are inadequate or lacking. Practical and effective vaccines that generate immunity to rabies or inhibit reproduction in specific species are needed. Basic dynamics, movement, and dispersal patterns of rabies vectors should be more fully investigated, particularly in relation to proposed disease control through oral vaccination or other techniques. Professional societies with diverse interests (ecology, mammalogy, wildlife biology, virology, and epidemiology) should collaborate and inform their members of activities in related fields through solicited papers and symposia. More complete species identification on animals, such as bats, submitted for testing should be completed by trained diagnostic laboratory personnel or through collaboration with mammalogists to correct potential laboratory personnel limitations with regard to taxonomic classification of animals submitted for diagnosis of rabies.

In addition to rabies surveillance of wildlife through conventional passive public health submissions, strategic application of active surveillance, such as at epizootic fronts and in areas implementing oral vaccination, will be critical. More effective use of available sources of data should be considered, including augmenting surveillance data collection at the state level. Information on specific geographic location and disease status of all animals submitted for testing should be reported and retained. Existing surveillance systems should be integrated within geographic information system databases, especially databases that would enable classification of habitat features. This would facilitate the understanding of the population dynamics and habitat relationships of reservoir species and potential spread of disease. Educational materials on rabies in wildlife and potential control measures need to be compiled and made available for widespread public distribution as requested by the public. The potential benefits of oral vaccination and other integrated control strategies should be thoroughly described for various major application strategies, such as suppression of local intensity of rabies, containment of an epizootic front, and proposed elimination of a terrestrial rabies variant. Research leading toward the development of practical contraceptives or related technology for managing wildlife populations should be encouraged and supported. Symposia that bring veterinarians, wildlife managers, and other stakeholders together for collaboration on management strategies should be conducted.

Authority for Management of Rabies in Wildlife

Timely and appropriate response concerning human or domestic animal exposure to rabies should be a local action. However, it is important for responses to be standardized and based on sound public health policy that requires protocols be developed at the state level, using national guidelines.[56] At the local level, a variety of agencies and individuals may be involved in managing exposure to rabies (eg, animal control officers, health department personnel, emergency room staff, and veterinarians). This multiagency involvement can be confusing for many citizens who may not know the responsible party to call in the event of an exposure. Such confusion can also lead to lack of coverage when clear lines of responsibility are not stated. Recommendations have been prepared by the National Association of State Public Health Veterinarians and the Advisory Committee on Immunization Practices, but these guidelines do not address specific logistic issues at the local level.

Recommendations—State and local task forces consisting of representatives of all involved agencies should be formed to make recommendations for improving communication and coordination at both levels. The health department should be the single authority at the local (city, county, or town) and state levels designated to establish protocols for the management of human exposure to wildlife and to ensure that protocols are followed. State health departments, cooperating with other state agencies (eg, agriculture, wildlife) and using information from recognized national authorities, should provide localities with guidelines and protocols, including those for the scientific rationale for managing wild and domestic animals that potentially expose humans or domestic animals. The public should be notified by various means as to appropriate contacts in the event of an exposure. The system of reporting exposures should be simple and should include 24-hour coverage for nights, weekends, and holidays. If local police or animal control dispatchers receive the information, it should be transmitted to the appropriate individual or agency (eg, health department, animal control, game warden) for response. In all instances, procedures should ensure that the health department is notified of any suspected exposure to rabies. Records should be kept of all potential exposures and eventual outcomes.

Management of Wildlife to Minimize Transfer of Disease

Throughout history, wild animals have been captured, moved, and released by human beings. In a report by Nielsen,[57] conservation, ecology, commerce, recreation, and humanitarian concerns were cited as the primary reasons for translocation of wildlife. Many benefits may be derived through translocation of wild animals, such as restoration of rare or endangered species and expansion of genetic variability of specific isolated populations. However, translocation of animals also has the potential for significant negative impact, particularly with regard to inadvertent transfer of pathogens. For example, there is evidence that the 1977 mid-Atlantic focus of the rabies epizootic in raccoons was the result of long-distance translocation of infected raccoons from the southeastern United States.[58-60] More recently, the coyote-dog variant of the rabies virus, previously known only from the United States-Mexico border,[4] was diagnosed in American Foxhounds in Alabama and Florida.[5] The cases were linked to commercial fox-chasing pens[61] that had stocked coyotes[5] and were contained. Intensive use of commercial enclosures created a need to restock animals and led to interstate commercial traffic in wild-caught foxes and coyotes.[61,62] In response, state regulations regarding fox-chasing enclosures and sale of live foxes and coyotes are rapidly evolving,[63] but compliance remains a problem.[62] In another recent incident, rabies was diagnosed in gray foxes transported from Texas to Montana. Genetic analysis revealed that the isolate was a gray fox variant found in west Texas.[3] Similar episodes have involved the translocation of bats from the United States to Europe.

Short-distance relocation of nuisance wildlife may also affect the local incidence of rabies. The most important reservoirs, such as raccoons, skunks, foxes, and various species of bats, are capable of living in close association with people, particularly where "suburbanization" results in adequate shelter and food. Nuisance wildlife are killed or captured and removed by property owners, private pest control operators, licensed commercial trappers, and municipal, state, or federal animal control or wildlife management personnel. Often, landowners express a strong desire that the animals be removed unharmed and transported elsewhere for release. Although relocation is often local, this transportation of animals may provide a mechanism for rabies to spread more rapidly into contiguous, susceptible populations or to surmount geographic or artificial immunologic barriers, such as those caused by oral vaccination of rabies in wildlife.

Recommendations—Stronger and more uniform federal and state wildlife regulations are necessary to prevent indiscriminate international, interstate, and intrastate movement and release of wild carnivores by private citizens. Effective enforcement of state wildlife regulations is necessary to deal with intrastate relocation of wild carnivores. Guidelines are critically needed for determining when nuisance wildlife should be euthanatized instead of being released. Regulations pertaining to the live release of nuisance animals that are vectors for rabies need to be more restrictive. Under the jurisdiction of the state's wildlife, agriculture, or public health agency, each state should have or develop regulations regarding the rehabilitation, capture, holding, sale, and release of wildlife, particularly the importation of wild-caught carnivores.

States with endemic rabies in a given species should develop regulations prohibiting—except under special permit—the assembly of live, wild animals of that species for any purpose including intra- or interstate sales. Violations of state regulations on import of wild animals should be prosecuted through a joint effort between the state and the US Fish and Wildlife Service, thereby activating the penalties associated with violations of the Lacey Act.[64]

Public education programs should be developed to explain public health risks and the need for regulations on relocation of wildlife. Information should address the zoonotic disease risks issues associated with translocation of wildlife. Stronger federal regulation of international animal importation, including the prohibition, quarantine, or restricted movement of exotic species capable of introducing or perpetuating nonindigenous Lyssaviruses, is required.

Implementation of Vaccination Programs for Wildlife

Use of ORV in the United States is restricted by the USDA to state or federally approved control programs targeting raccoons, with additional applications underway in Texas targeting coyotes and gray foxes. Parenteral vaccines have not been licensed for use in wildlife. Use of ORV should be reserved for large-scale attempts to eliminate or reduce the impact of an outbreak of rabies in wildlife or to limit entry of rabies from wildlife into an area; ORV should not be used for the vaccination of individual animals. Currently, there is no officially delegated lead agency to monitor or evaluate the use of ORV once they are fully licensed.

Recommendations—A national strategy should be formulated for the use of ORV in wildlife, and a federal agency should be designated to lead wildlife vaccination efforts. A single agency within each state should be designated to coordinate rabies vaccination programs in consultation with the other involved agencies. Oral vaccination programs should be optimized through investigations of various bait densities and distribution methods. The CDC should provide technical laboratory and logistic assistance in the conduct, coordination, and surveillance evaluation of state programs, including communication and coordination with other participating state and federal agencies. The USDA should assist in implementation of control programs. Universities and other groups could play various roles, including research, evaluation, and technical support; however, these roles should be secondary to the activities of state and federal agencies. State authorities from public health agencies and either the designated public health veterinarian or the state veterinarian from agriculture departments should have ultimate responsibility for the conduct, supervision, coordination, and termination of wildlife vaccination programs in their respective states. These activities should be coordinated between state departments of agriculture and wildlife. This effort can be expedited by the formation of an interdepartmental task force or committee representing at least those 3 agencies responsible for public health, agriculture, and wildlife. Other potential members for such a task force include private and academic veterinary and human medical practitioners and biomedical researchers.

Since the meeting of the 1995 working group, measurable, but somewhat limited, progress has been made toward control of rabies in wildlife. More areas are using ORV since the vaccine has become licensed. Further westward advancement of rabies in raccoons appears to have been stalled by a considerable ORV effort in the first affected Ohio counties, adjacent to Pennsylvania. However, the recent advancement of rabies northward from New York to eastern Ontario, despite prevention measures in the area (ORV in New York and TVR in Ontario), exemplifies the weakness of current control methods and the lack of guidelines toward efficacious application. Application of ORV in Texas has restricted the progression of rabies in coyotes and gray foxes. Clearly, ORV and other management methods are currently novel tools in the prevention and control of rabies in the United States. For these control methods to become practical, numerous aspects of the various techniques will require additional development and evaluation. Economic analysis and field assessment is in progress.

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Public Veterinary Medicine: Public Health

Rabies surveillance in the United States during 2008

Jesse D. Blanton, MPH; Kis Robertson, DVM, MPH; Dustyn Palmer, BA; Charles E. Rupprecht, VMD, PhD

Summary—During 2008, 49 states and Puerto Rico reported 6,841 cases of rabies in animals and 2 cases in humans to the CDC, representing a 3.1% decrease from the 7,060 cases in animals and 1 case in a human reported in 2007. Approximately 93% of the cases were in wildlife, and 7% were in domestic animals. Relative contributions by the major animal groups were as follows: 2,389 (34.9%) raccoons, 1,806 (26.4%) bats, 1,589 (23.2%) skunks, 454 (6.6%) foxes, 294 (4.3%) cats, 75 (1.1%) dogs, and 59 (0.9%) cattle. Compared with numbers of cases reported in 2007, numbers of cases reported in 2008 increased among cats, cattle, and skunks and decreased among dogs, raccoons, bats, and foxes. Numbers of rabid raccoons reported during 2008 decreased in 11 of the 20 eastern states where raccoon rabies was enzootic; overall number of rabid raccoons reported decreased by 8.6% during 2008, compared with 2007.

On a national level, the number of rabies cases involving skunks increased by 7.7% during 2008, compared with the number reported in 2007; this was the first increase in the number of reported rabid skunks since 2006. The total number of cases of rabies reported nationally in foxes decreased 1.7% in 2008, compared with 2007. The 1,806 cases of rabies reported in bats represented a 6.7% decrease, compared with the number reported in 2007. One case of rabies in a dog imported from Iraq was reported at a quarantine station in New Jersey during 2008. Follow-up of potentially exposed animals in the same shipment did not reveal any secondary transmission. The United States remained free from dog-to-dog transmission of canine rabies virus variants. Total number of rabid dogs reported decreased 19.4% in 2008, compared with 2007.

Two human rabies cases were reported from California and Missouri during 2008. The California case involved a recent immigrant from Mexico and was attributed to a newly identified rabies virus variant most likely associated with Mexican free-tailed bats. The case in Missouri was attributed to a rabies virus variant associated with eastern pipistrelle and silver-haired bats.

The present report provides an update on rabies epidemiology and events in the United States during 2008. Summaries of 2008 surveillance data for Canada and Mexico are also provided because of their common borders with the United States and the frequent travel between the United States and these countries. A brief preliminary update on cases of rabies and other related activities reported to the CDC during 2009 is also included.

As is the case in many developed countries, wild animals accounted for the majority (93%) of all rabies cases in the United States reported to the CDC during 2008. The most frequently reported rabid wildlife were raccoons, bats, skunks, and foxes; however, their relative proportions have continued to fluctuate over the years owing to epizootics of rabies among animals infected with various distinct rabies virus variants.[1]

Rabies virus infections involving terrestrial animals in the United States occur in geographically definable regions where virus transmission is primarily between members of the same species. Spillover infection from these species to other animals occurs but rarely initiates sustained transmission in other species. Once established, enzootic virus transmission within a species can persist regionally for decades or longer.

The spatial boundaries of enzootic rabies in reservoir species are temporally dynamic (Figure 1), and affected areas may expand and contract as a result of virus transmission and animal population interactions.[2,3] Population increases and emigration result in expansion of enzootic areas, whereas natural barriers, such as mountain ranges and bodies of water, may sustain lower population densities or restrict animal move-

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ments, slowing the spread of rabies. Unusual animal dispersal patterns and human-mediated translocation of infected animals have resulted in more rapid or unexpected introduction of rabies into new areas.[1-6]

The canine rabies virus variant, which is responsible for dog-to-dog rabies transmission, was reintroduced in coyotes in the United States in the late 1980s, but, following > 10 years of oral vaccination, has again been eliminated.[7-10] An ongoing analysis of the phylogenetics of circulating terrestrial rabies virus variants has suggested that canine rabies virus variants were the probable origins of several circulating wildlife rabies virus variants of foxes (Texas and Arizona) and skunks (California and north central United States). This is likely representative of a long process that began with the introduction of canine rabies during colonization of the Americas followed by spillover and adaptation of Old World canine rabies virus variants to New World wildlife species, which have maintained an independent sylvatic circulation of canine origin rabies virus variants.[11]

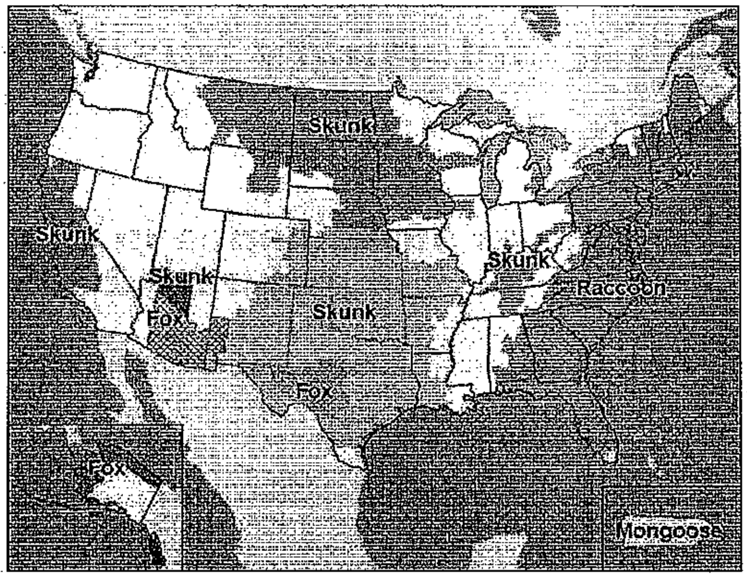

Figure 1—Distribution of major rabies virus variants among wild terrestrial animal reservoirs in the United States and Puerto Rico, 2008.

Following translocation of rabid raccoons (*Procyon lotor*) from an enzootic area in the southeastern United States to the mid-Atlantic region, raccoon rabies spread rapidly and has become enzootic in all of the eastern coastal states as well as in Alabama, Ohio, Pennsylvania, Tennessee, Vermont, and West Virginia. Three different rabies virus variants are responsible for disease in skunks (primarily *Mephitis mephitis*) in California and the north central and south central United States. In Alaska, a long-standing reservoir for rabies virus exists in arctic and red foxes (*Alopex lagopus* and *Vulpes vulpes*, respectively). Two different rabies virus variants are present in geographically limited populations of gray foxes (*Urocyon cinereoargenteus*) in Arizona and Texas. On the island of Puerto Rico, another wildlife rabies reservoir exists in mongooses (*Herpestes javanicus*).[12,13]

Distribution of an oral vaccinia–rabies glycoprotein recombinant vaccine targeting raccoons in the eastern United States[14-16] and gray foxes and coyotes (*Canis latrans*) in Texas[10] has shown promise as an important adjunct to traditional rabies control methods (ie, parenteral vaccination of domestic animals). Biologics used in oral vaccination programs contain live replicating virus, and the unintentional exposure of nontarget species, including humans, must be minimized and monitored.[17-19]

There are multiple, independent reservoirs of rabies virus in several species of insectivorous bats, with distribution patterns overlaying the distribution of rabies virus variants maintained in terrestrial mammals. Rabies virus transmission among bats appears to be primarily intraspecific, and distinct virus variants can be identified and associated with different bat species. In contrast to maintenance cycles in terrestrial animals, however, the greater mobility of bats precludes definitive range-mapping of different variants, other than the geographic ranges of the implicated host bat species. Because bat species known to be reservoirs for rabies virus are found in all areas of the continental United States, every state except Hawaii is considered enzootic for rabies.

Various public health activities, including vaccination of companion animals, vaccination programs targeting wildlife, and ongoing education programs, have contributed to the reduction in transmission of rabies virus from terrestrial animals to humans.[20] As a result, most cases of rabies in humans have resulted from infection with rabies virus variants associated with bats.[21,22] Rabies control in bats by conventional methods is difficult, and preventing infection with bat-associated rabies virus variants in humans is further complicated by the frequent absence of documented exposure histories involving a bat bite.

Reporting and Analysis

The number of reported cases of rabies represents only a fraction of the total cases that occur each year. Many rabid animals are never observed and therefore go undetected and untested.[23] The predominantly passive nature of public health and veterinary rabies surveillance programs and the lack of accurate estimates of animal populations mean that incidence and prevalence of rabies cannot be accurately determined for most species. Existing public health reporting systems were not designed for transmission of data involving diseases in animal populations and often lack designated fields for reporting vital information such as animal species.[24,25] Furthermore, laboratory-based reporting of rabies cases to the CDC is complicated by the presence of multiple laboratories that perform rabies diagnostic testing in some states (eg, public health, agricultural, and veterinary pathology laboratories).

During 2008, 8 states (Georgia, Massachusetts, Maryland, Michigan, North Dakota, Virginia, Vermont, and West

Virginia) transmitted testing data electronically through the use of the updated Public Health Laboratory Isolate Surveillance system, which leverages the Public Health Informatics Network–Messaging System to securely transmit text files in a defined messaging format to a CDC database. With the creation of this electronic, laboratory-based reporting system, the CDC database for rabies surveillance data has been restructured to allow collection of data for individual animals, so that additional data elements can be submitted on each animal, as opposed to the historical reporting of aggregate counts by species and county. The updated Public Health Laboratory Isolate Surveillance system provides a stopgap solution for electronic reporting of animal rabies testing data while standards-based messaging guides for animal rabies reporting are being developed at the federal and state levels. The system's relative ease of use and independence from specific laboratory information systems (most database systems can export data to text files) make its implementation in non–public health laboratories feasible. Additional information provided voluntarily by some state health departments during 2008 included sex, age, and vaccination status of rabid animals; human and animal exposures to rabid animals; coordinate or street address of collection; and variant typing information.

To facilitate consistent reporting from states that do not use the Public Health Laboratory Isolate Surveillance system, all states and territories are requested to submit finalized data directly to the Poxvirus and Rabies Branch of the CDC. In animals suspected of having rabies, a diagnosis was made by detecting rabies viral antigen in brain material submitted to state laboratories by means of a direct immunofluorescent antibody test, as described.[26] Virus isolation in neuroblastoma cell cultures or in mice, nucleic acid detection via a reverse transcriptase PCR assay, and sequencing and genetic analysis were used to confirm the diagnosis in some cases. This year, CDC also requested direct reporting of testing activity by USDA Wildlife Services field biologists who were using a direct rapid immunohistochemistry test[27] for enhanced rabies surveillance, and information was provided for 7,088 samples, representing 5.8% of all samples reportedly tested during 2008. All samples for which the direct rapid immunohistochemistry test provided positive or indeterminate results and 10% of samples for which the test provided negative results were submitted to the CDC for confirmation by means of the direct fluorescent antibody test.

Between January 1 and December 31, 2008, all 50 states, New York City, the District of Columbia, and Puerto Rico reported numbers of cases of animal rabies to the CDC. All states also provided data on total rabies diagnostic testing activity during 2008. However, county of origin was not reported for animals from the state of Oklahoma for which test results were negative. A total of 121,728 animals were reportedly tested in the United States during 2008, accounting for a 0.7% increase in the number of animals tested, compared with 2007.

For rabies cases involving most terrestrial mammals, state public health laboratories generally report the common name of affected animals, with affected animals typically identified to the level of genus and often to the level of species. However, for cases involving bats, affected animals are frequently identified only to the level of taxonomic order (ie, Chiroptera) because not all public health laboratories have the capacity to speciate bats, even though they are encouraged to do so.

All year-end totals included in the present report were confirmed through e-mail or telephone conversations with state or territorial health department officials. Data from Canada were obtained from the Terrestrial Animal Health Division, Canadian Food Inspection Agency, and data from Mexico were obtained from the Pan American Health Organization Epidemiological Information System.[a]

State health authorities have different requirements for submission of specimens for rabies testing; therefore, intensity of surveillance varies. To better estimate regional trends, determine the rigor of surveillance efforts, and identify possible biases, states are encouraged to submit denominator data (ie, data for animals tested, but for which results of direct fluorescent antibody testing were negative) by species, county, and temporal occurrence. Calculations of percentages of positive test results are based on the total number of animals tested for rabies. Because most animals submitted for testing are selected because of abnormal behavior or obvious signs of illness, percentages of tested animals with positive results in the present report are not representative of the incidence of rabies in the general population. Further, because of differences in protocols and submission rates among species and states, comparison of percentages of animals with positive results between species or states is inappropriate. For comparison of historical rates, data from states lacking total submission data were excluded from calculations.

Geographic areas for various rabies virus reservoirs in the United States were produced by aggregating data from 2004 through 2008. County boundaries where cases were reported in the reservoir species over this period were dissolved with a geographic information system[b] to produce a single polygon representing the distribution of a specific rabies virus variant. Reservoir maps are an estimate of the relative distribution of each major terrestrial rabies virus variant maintained by a particular reservoir species. Owing to the

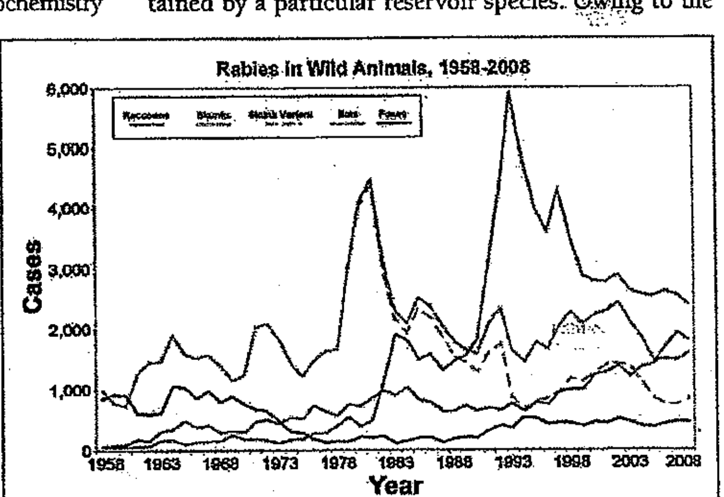

Figure 2—Cases of rabies in wild animals in the United States, by year and species, 1958 to 2008.

paucity of samples tested at some localities and a lack of antigenic typing or genetic sequencing where reservoirs meet, defining precise viral fronts is difficult. Geographic location was provided only to the county level, and maps represent cases at this jurisdictional level. Because of the positive skew of the data, a geometric classification scheme was used for choropleth maps used to display number of animals tested; pro-

Table 1—Cases of rabies in the United States, by state and category, during 2008.

				Domestic animals						Wild animals									
State (city)	Total cases	Domestic	Wild	Cats	Cattle	Dogs	Horses/ mules	Sheep/ goats	Other domestic*	Raccoons	Bats	Skunks	Foxes	Other wild†	Rodents and lagomorphs‡	Humans	% Positive 2008	2007 cases	Change (%)
AK	15	0	15	0	0	0	0	0	0	0	0	0	15	0	0	0	31.2	45	−66.67
AL	84	2	82	0	0	1	1	0	0	51	17	0	11	3[b]	0	0	3.8	80	5.00
AR	49	5	44	0	0	4	0	1	0	0	5	39	0	0	0	0	5.1	33	48.48
AZ	182	2	180	0	0	1	1	0	0	0	89	57	21	13[c]	0	0	6.6	159	14.47
CA	179	1	177	1	0	0	0	0	0	0	137	31	9	0	0	1	2.5	188	−4.79
CO	65	1	64	1	0	0	0	0	0	1	44	19	0	0	0	0	6.9	56	16.07
CT	202	11	191	11	0	0	0	0	0	109	40	32	6	3[d]	1[t]	0	7.7	219	−7.76
DC	49	6	43	6	0	0	0	0	0	28	12	0	3	0	0	0	10.9	43	13.95
DE§	21	7	13	6	0	0	1	0	0	7	4	0	1	0	1[u]	0	8.4	11	90.91
FL	151	11	140	9	0	0	2	0	0	95	20	2	20	3[e]	0	0	3.9	128	17.97
GA	389	24	365	15	1	6	1	0	1[a]	235	25	62	36	7[f]	0	0	14.8	301	29.24
HI	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0.0	0	0.00
IA	27	10	17	8	1	1	0	0	0	0	11	6	0	0	0	0	1.7	31	−12.90
ID	10	0	10	0	0	0	0	0	0	0	10	0	0	0	0	0	2.0	12	−16.67
IL	103	0	103	0	0	0	0	0	0	0	103	0	0	0	0	0	1.7	113	−8.85
IN	13	0	13	0	0	0	0	0	0	0	13	0	0	0	0	0	0.8	13	0.00
KS	67	13	54	11	0	0	2	0	0	0	6	48	0	0	0	0	5.5	110	−39.09
KY	46	9	37	1	0	6	2	0	0	0	14	23	0	0	0	0	3.5	20	130.00
LA	6	0	6	0	0	0	0	0	0	0	3	3	0	0	0	0	0.9	6	0.00
MA	154	18	136	15	0	1	1	1	0	62	19	44	8	1[g]	2[v]	0	5.3	152	1.32
MD	420	27	393	21	2	1	2	1	0	271	33	41	37	0	11[w]	0	9.2	431	−2.55
ME	65	0	65	0	0	0	0	0	0	33	8	21	3	0	0	0	9.2	86	−24.42
MI	79	1	78	1	0	0	0	0	0	0	70	6	2	0	0	0	2.1	210	−62.38
MN	70	9	61	2	4	3	0	0	0	0	33	28	0	0	0	0	2.4	40	75.00
MO	66	1	64	0	0	0	1	0	0	0	58	6	0	0	0	1	2.1	38	73.68
MS	7	0	7	0	0	0	0	0	0	0	7	0	0	0	0	0	2.1	3	133.33
MT	14	1	13	0	0	1	0	0	0	0	11	2	0	0	0	0	3.3	23	−39.13
NC	474	25	449	18	3	3	0	1	0	270	18	93	60	7[h]	1[x]	0	12.5	474	0.00
ND	34	14	20	5	4	4	1	0	0	1	1	18	0	0	0	0	7.8	30	13.33
NE	43	7	36	1	4	0	2	0	0	0	10	25	1	0	0	0	4.0	31	38.71
NH	59	3	56	1	0	0	0	2	0	28	3	19	6	0	0	0	9.7	54	9.26
NJ	285	17	268	15	0	2	0	0	0	155	57	44	6	1[i]	5[y]	0	8.5	283	0.71
NM	25	2	23	1	0	1	0	0	0	0	0	6	17	0	0	0	5.8	17	47.06
NV	16	0	16	0	0	0	0	0	0	0	16	0	0	0	0	0	4.0	9	77.78
NY	496	31	465	23	6	1	1	0	0	262	112	63	20	3[j]	5[z]	0	5.6	512	−3.13
NYC	19	1	18	1	0	0	0	0	0	9	2	7	0	0	0	0	2.5	47	−59.57
OH	64	0	64	0	0	0	0	0	0	5	55	3	0	1[k]	0	0	1.5	86	−25.58
OK	43	11	32	2	6	2	1	0	0	0	2	29	0	1[l]	0	0	3.8	78	−44.87
OR	13	0	13	0	0	0	0	0	0	0	13	0	0	0	0	0	5.1	12	8.33
PA	431	60	371	53	3	3	0	1	0	228	43	71	25	2[m]	2[aa]	0	4.6	439	−1.82
PR	58	16	42	3	1	11	1	0	0	0	0	0	0	42[n]	0	0	28.3	47	23.40
RI	34	2	32	1	0	0	1	0	0	9	7	12	4	0	0	0	7.4	45	−24.44
SC	166	6	160	3	2	1	0	0	0	81	10	34	34	1[o]	0	0	7.2	162	2.47
SD	24	4	20	0	2	0	2	0	0	0	4	16	0	0	0	0	3.5	27	−11.11
TN	128	6	122	2	0	3	1	0	0	28	18	69	7	0	0	0	4.9	132	−3.03
TX	1,022	45	977	15	9	15	4	2	0	16	548	393	14	6[p]	0	0	7.1	969	5.47
UT	14	0	14	0	0	0	0	0	0	0	14	0	0	0	0	0	2.5	16	−12.50
VA	622	48	574	34	6	4	2	2	0	310	22	158	78	1[q]	5[bb]	0	14.4	730	−14.79
VT	75	1	74	0	1	0	0	0	0	41	3	25	3	1[r]	1[cc]	0	13.3	165	−54.55
WA	17	0	17	0	0	0	0	0	0	0	17	0	0	0	0	0	3.1	22	−22.73
WI	24	0	24	0	0	0	0	0	0	0	24	0	0	0	0	0	1.1	26	−7.69
WV	96	13	83	8	4	0	0	1	0	54	3	18	7	1[s]	0	0	5.1	77	24.68
WY	28	0	28	0	0	0	0	0	0	0	12	16	0	0	0	0	4.0	19	47.37
Total	6,843	471	6,369	294	59	75	30	12	1	2,389	1,806	1,589	454	97	34	2	5.62	7,060	−3.07
% 2008	100.00	6.90	93.07	4.30	0.86	1.10	0.44	0.18	0.01	34.91	26.39	23.22	6.63	1.42	0.50	0.03			
% Pos 2008	5.62	0.78	10.47	0.95	4.97	0.28	3.02	2.60	0.30	14.48	5.96	26.55	24.89	3.49	0.99	—			
Total 2007	7,060	469	6,590	262	57	93	41	13	3	2,549	1,935	1,476	462	118	50	1			
% Change	−3.07	0.43	−3.35	12.21	3.51	−19.35	−26.83	−7.69	−66.67	−6.28	−6.67	7.66	−1.73	−17.80	−32.00	100.00			

*Other domestic includes: [a]1 llama. †Other wild includes: [b]1 bobcat, 2 coyotes; [c]7 bobcats, 1 coati, 1 cougar, 4 coyotes; [d]1 coyote, 2 deer; [e]3 bobcats; [f]5 bobcats, 2 coyotes; [g]1 bobcat; [h]3 bobcats, 3 coyotes, 1 opossum; [i]1 opossum; [j]1 coyote, 2 deer; [k]1 coyote; [l]1 bobcat; [m]1 bobcat, 1 deer; [n]42 mongooses; [o]1 opossum; [p]6 coyotes; [q]1 opossum; [r]1 otter; [s]1 deer. ‡Rodents and lagomorphs include: [t]1 groundhog; [u]1 rabbit; [v]2 groundhogs; [w]11 groundhogs; [x]1 beaver; [y]5 groundhogs; [z]4 groundhogs, 1 rabbit; [aa]2 groundhogs; [bb]5 groundhogs; [cc]1 groundhog. §One rabid animal reported from Delaware without species information.

% Pos = (Total number positive/total number tested) X 100. — = Not calculated.

portional symbols were used to display reported cases by county. All maps were constructed with the Albers equal-area conic projection to minimize areal distortion over the United States.

Rabies in Wild Animals

Wild animals accounted for 6,369 (93.1%) of the 6,841 reported cases of rabies in 2008 (Figure 2). This number represented a 3.4% decrease from the 6,590 cases reported in 2007 (Table 1). Raccoons continued to be the most frequently reported rabid wildlife species (34.9% of all animal cases during 2008), followed by bats (26.4%), skunks (23.2%), foxes (6.6%), and other wild animals, including rodents and lagomorphs (1.9%). Numbers of reported cases in raccoons, bats, and foxes decreased 6.3%, 6.7%, and 1.7%, respectively, compared with 2007 totals. Reported cases in skunks increased 7.7%, compared with 2007. Seasonal trends for wildlife species were similar to previous years, with peaks in reported cases of raccoons, skunks, and foxes in March and May, with a second higher peak among raccoons and skunks in August and September. Reports of rabid bats had a single peak in August.

Raccoons—The 2,389 cases of rabies in raccoons reported in 2008 represented a continued declining trend since the last increase reported in 2006 (Table 1). Overall, the percentage of raccoons with positive test results has also decreased, from 17.7% in 2007 to 14.5% in 2008. Decreases of ≥ 50% in the numbers of rabid raccoons during 2008 were reported by 3 of the 20 eastern states where raccoon rabies is enzootic (ie, Vermont, 60.2% decrease from 2007 [103 cases] to 2008 [41 cases]; Rhode Island, 57.1% decrease from 2007 [21 cases] to 2008 [9 cases]; and Ohio, 54.5% decrease from 2007 [11 cases] to 2008 [5 cases]) and by New York City (77.5% decrease from 2007 [40 cases] to 2008 [9 cases]; Figure 3). Delaware (75.0% increase from 2007 [4 cases] to 2008 [7 cases]) was the only state to report a ≥ 50% increase in the number of rabid raccoons. States in the northeastern and mid-Atlantic focus of the raccoon rabies epizootic accounted for 67.4% (1,611 cases; 13.7% decrease) of the 2,389 total rabies cases in raccoons during 2008. The southeastern states of Alabama, Florida, Georgia, North Carolina, South Carolina, and Tennessee reported 31.8% (760 cases; 1.6% decrease) of the total cases in raccoons. Excluding Tennessee and Ohio, where skunk rabies is also present, states where raccoon rabies was the only terrestrial rabies virus variant reported 97.9% (2,338/2,389) of all documented cases of rabies in raccoons and accounted for 62.7% (4,292/6,841) of the national total of rabid animals (76.1% [3,834/5,035] of total cases in terrestrial animals).

Rabid raccoons reported by Texas (n = 16), North Dakota (1), and Colorado (1) were presumably the result of spillover infection from local terrestrial reservoirs. Fourteen of the cases in Texas were attributed to the south central skunk rabies virus variant (2 cases were untyped). The case in Colorado was attributed to the south central skunk rabies virus variant. The virus variant in the case in North Dakota was untyped.

Bats—The 1,806 cases of rabies reported in bats during 2008 represented a decrease of 6.7%, compared with the number reported in 2007. Total percentage of tested bats with positive results also decreased from 6.4% in

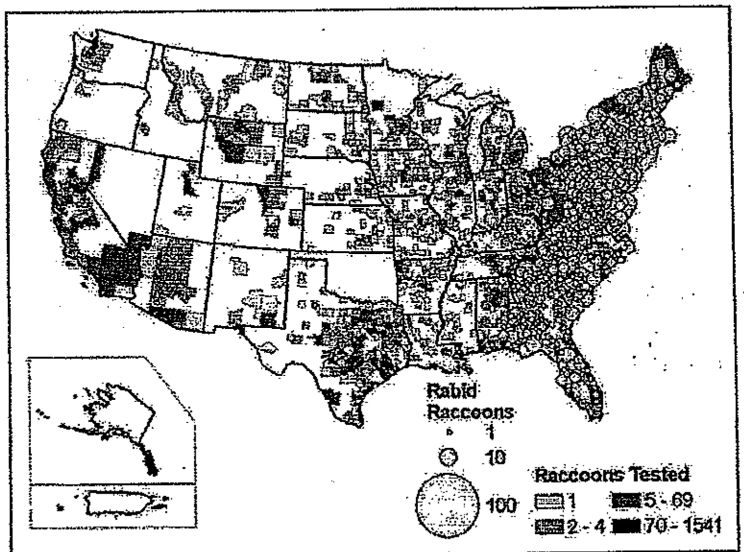

Figure 3—Reported cases of rabies in raccoons, by county, 2008.

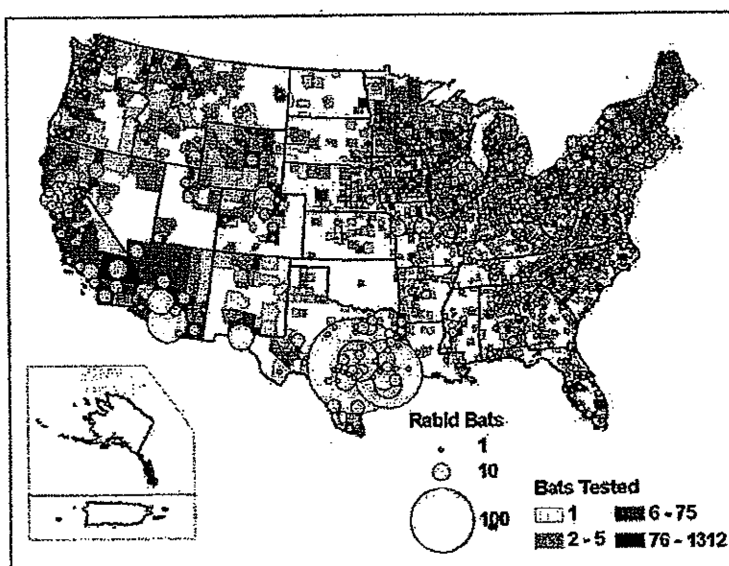

Figure 4—Reported cases of rabies in bats, by county, 2008.

2007 to 6.0% in 2008. Rabies in bats was widely distributed throughout the United States, with cases reported from 47 of the 48 contiguous states (Figure 4). Alaska, Hawaii, New Mexico, and Puerto Rico did not report any cases of bat rabies during 2008. Four states reported > 100 cases of rabies in bats, and these 4 states accounted for nearly half of the reported cases of rabies in bats during 2008 (Texas, 548 cases [30.3%]; California, 137 cases [7.6%]; New York, 112 cases [6.2%]; and Illinois, 103 cases [5.7%]). Nine states (Idaho, Illinois, Indiana, Mississippi, Nevada, Oregon, Utah, Washington, and Wisconsin) reported rabies in bats but not in terrestrial mammals. Of the bats infected with rabies virus, 26.3% (475/1,806) were identified beyond the taxonomic level of order (18 to the level of genus and 457 to the level of species). Among bats identified beyond the taxonomic level of order, 63.8% (303) were the big brown bat (*Eptesicus fuscus*), 10.1% (48) were the Brazilian (Mexican) free-tailed bat (*Tadarida brasiliensis*), 4.4% (21) were the hoary bat (*Lasiurus cinereus*), 4.2% (20) were the red bat (*Lasiurus borealis*), 4.0% (19) were the western pipistrelle (*Pipistrellus hesperus*), 3.4% (16) were the little brown bat (*Myotis lucifugus*), 1.9% (9) were the silver-haired bat (*Lasionycteris noctivagans*), 1.3% (6) were the pallid bat (*Antrozous pallidus*), 0.6% (3) were the long-legged myotis (*Myotis volans*), 0.4% (2) were the California myotis (*Myotis californicus*), 0.4% (2) were the northern long-eared myotis (*Myotis septentrionalis*), 0.4% (2) were the Yuma myotis (*Myotis yumanesis*), 0.4% (2) were the western yellow bat (*Lasiurus xanthinus*), 0.4% (2) were the big free-tailed bat (*Nyctinomops macrotis*), 0.2% (1) was the evening bat (*Nycticeius humeralis*), and 0.2% (1) was the Seminole bat (*Lasiurus seminolus*). Unspeciated bats of the genus *Myotis* (18/475) accounted for the remaining rabid bats and contributed 3.8% to the total number of bats identified beyond the taxonomic level of order.

Skunks—The 1,589 reported cases of rabies in skunks (mainly *M mephitis*) in 2008 represented a 7.7% increase from the number reported in 2007 (Figure 5; Table 1). However, the total percentage of tested skunks with positive results was the same during 2008 (26.6%) as during 2007. Eleven of the 24 states where a skunk rabies virus variant was enzootic reported a ≥ 50% increase in the number of rabid skunks during 2008 (ie, Kentucky, 475% increase from 2007 [4 cases] to 2008 [23 cases]; Colorado, 375% increase from 2007 [4 cases] to 2008 [19 cases]; Arizona, 338% increase from 2007 [13 cases] to 2008 [57 cases]; Wyoming, 300% increase from 2007 [4 cases] to 2008 [16 cases]; New Mexico, 200% increase from 2007 [2 cases] to 2008 [6 cases]; Louisiana, 200% increase from 2007 [1 case] to 2008 [3 cases]; Nebraska, 92% increase from 2007 [13 cases] to 2008 [25 cases]; Arkansas, 70% increase from 2007 [23 cases] to 2008 [39 cases]; North Dakota, 64% increase from 2007 [11 cases] to 2008 [18 cases]; Minnesota, 56% increase from 2007 [18 cases] to 2008 [28 cases]; and Missouri, 50% increase from 2007 [4 cases] to 2008 [6 cases]). Illinois, Indiana, and Wisconsin reported no rabies in skunks during 2008. Indiana reported a single case of rabies in a skunk in 2007, Illinois has not reported a case of rabies in a skunk since 2005, and Wisconsin has not reported a case of rabies in a skunk since 2006. Montana reported a 67% decrease in the number of rabid skunks from 2007 (6 cases) to 2008 (2 cases).

States in which the raccoon rabies virus variant is enzootic (excluding Tennessee, where skunks are the predominant reservoir) reported 47.1% (749/1,589) of the cases of rabies in skunks, most of which were presumably the result of spillover infection from raccoons. This was a slight

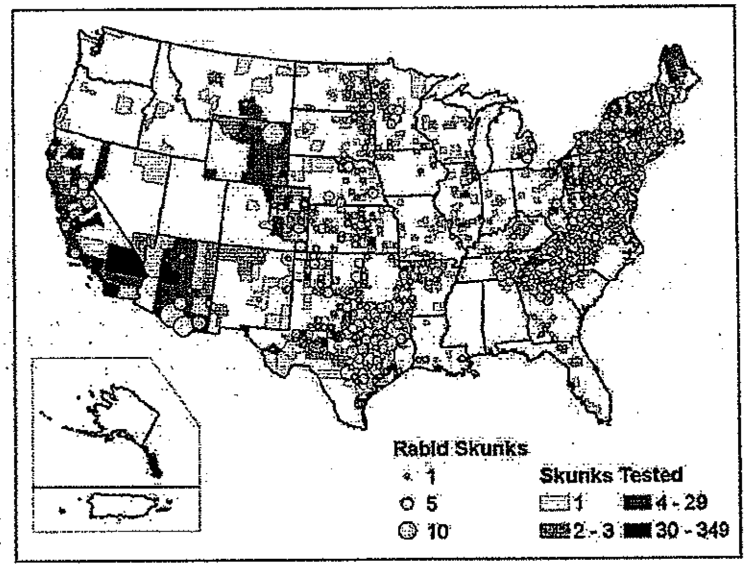

Figure 5—Reported cases of rabies in skunks, by county, 2008.

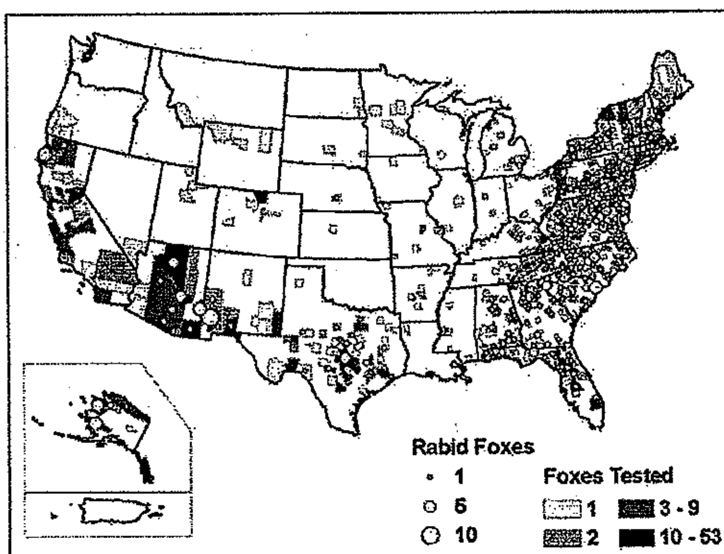

Figure 6—Reported cases of rabies in foxes, by county, 2008.

decrease from the proportion of rabid skunks presumably infected with the raccoon rabies virus variant in previous years. Among the 19 states where the raccoon rabies virus variant is the predominant terrestrial reservoir of rabies, 4 states (South Carolina, 143% increase from 2007 [14 cases] to 2008 [34 cases]; Georgia, 77% increase from 2007 [35 cases] to 2008 [62 cases]; New Jersey, 57% increase from 2007 [28 cases] to 2008 [44 cases]; and West Virginia, 50% increase from 2007 [12 cases] to 2008 [18 cases]) and New York City (133% increase from 2007 [3 cases] to 2008 [7 cases]) reported ≥ 50% increases in the number of rabid skunks. Rhode Island reported more rabid skunks than raccoons for the first time since 2005.

Foxes—Foxes (mainly *A lagopus*, *U cinereoargenteus*, or *V vulpes*) accounted for 6.6% of all cases of rabies in animals reported in 2008 (Table 1). The 454 cases of rabies in foxes represented a 1.7% increase from 2007. The percentage of tested foxes with positive results decreased from 28.4% in 2007 to 24.9% in 2008. Most cases of rabies in foxes (368 [81.0%]) were reported by states affected predominantly by the raccoon rabies virus variant (Figure 6). Ten states (Connecticut, 500% increase from 2007 [1 case] to 2008 [6 cases]; West Virginia, 250% increase from 2007 [2 cases] to 2008 [7 cases]; New Hampshire, 200% increase from 2007 [2 cases] to 2008 [6 cases]; Massachusetts, 167% increase from 2007 [3 cases] to 2008 [8 cases]; Georgia, 100% increase from 2007 [18 cases] to 2008 [36 cases]; Rhode Island, 100% increase from 2007 [2 cases] to 2008 [4 cases]; New Mexico, 78% increase from 2007 [9 cases] to 2008 [16 cases]; and California, 50% increase from 2007 [6 cases] to 2008 [9 cases]) and the District of Columbia (200% increase from 2007 [1 case] to 2008 [3 cases]) reported a ≥ 50% increase in the number of rabid foxes, compared with 2007. Nebraska and Tennessee reported no cases of rabies in foxes during 2007 but reported 1 and 7 cases, respectively, during 2008.

Other wild animals—Puerto Rico reported 42 rabid mongooses (*H javanicus*) during 2008, a 31% increase from the 32 cases reported in 2007 (Table 1). Other wildlife in which rabies was reported included 31 groundhogs (*Marmota monax*), 22 bobcats (*Lynx rufus*), 20 coyotes (*C latrans*), 6 white-tail deer (*Odocoileus virginianus*), 4 opossums (*Didelphis virginiana*), 2 rabbits (species not identified), 1 beaver (*Castor canadensis*), 1 coati (*Nasua narica*), 1 cougar (*Puma concolor*), and 1 river otter (*Lontra canadensis*). All cases of rabies in rodents and lagomorphs were reported by states in which rabies is enzootic in raccoons.

For 17 of the 20 coyotes positive for rabies, the variant was typed. Variant information was not reported for cases in Connecticut (n = 1) and Georgia (2). All rabid coyotes for which variant typing results were available were infected with the predominant terrestrial rabies virus variant for the geographic region where the animal was found (4 infected with the Texas gray fox rabies virus variant, 7 infected with the raccoon rabies virus variant, 2 infected with the south central skunk rabies virus variant, and 4 infected with the Arizona gray fox virus variant).

Rabies in Domestic Animals

Domestic species accounted for 6.9% of all rabid animals reported in the United States in 2008 (Table 1). The number of rabid domestic animals reported in 2008 (471) represented a 0.5% increase from the total reported in 2007 (Figure 7). Cases of rabies reported in dogs, horses, and sheep and goats decreased by 19.4%, 26.8%, and 7.7%, respectively, while cases of rabies reported in cats and cattle increased 12.2% and 3.5%, respectively. Pennsylvania reported the largest number of rabid domestic animals (60 cases), followed by Virginia (48), Texas (45), New York (31), Maryland (27), and North

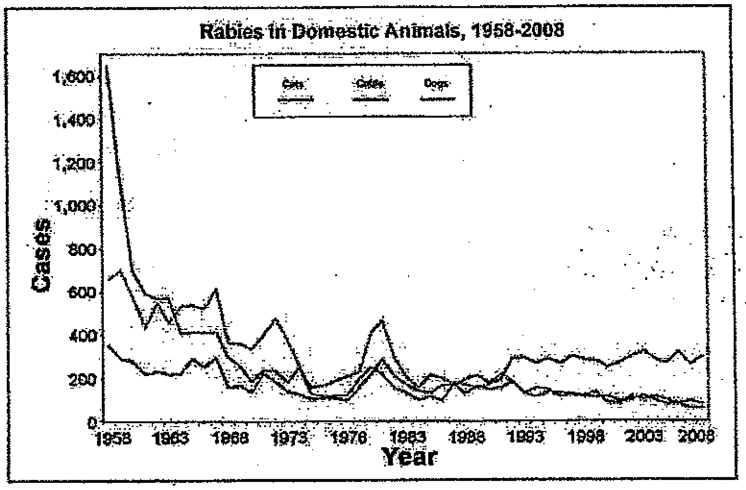

Figure 7—Cases of rabies in domestic animals in the United States, by year, 1958 to 2008.

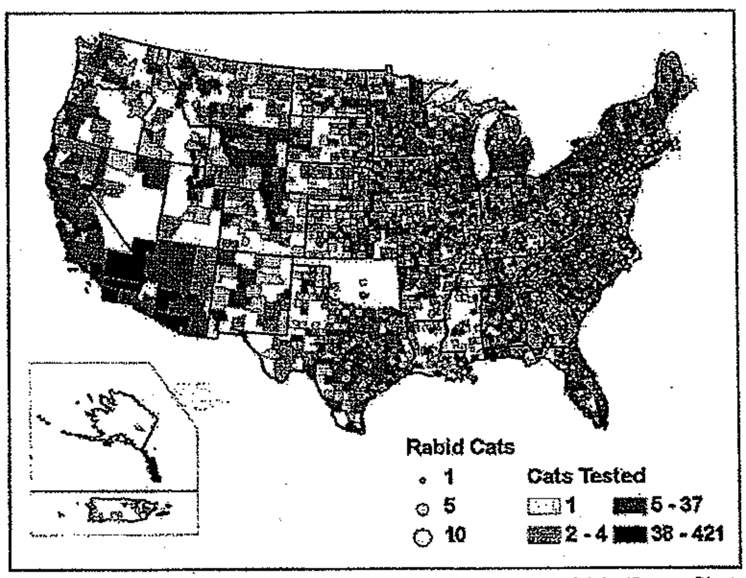

Figure 8—Reported cases of rabies in cats, by county and municipio (Puerto Rico), 2008.

Carolina (25). Seasonal distribution for reporting of rabies in domestic animals was similar to that for previous years. Reported cases of rabies in cats had a slight peak during June and July. Reported cases of rabies in cattle and dogs do not show any strong seasonal patterns.

Cats—The number of cases of rabies reported in cats was nearly 4 times the number reported for dogs and 5 times the number reported for cattle. Most (82.3%) of the 294 cases of rabies in cats were reported from states in which the raccoon rabies virus variant was present (Figure 8). Remaining cases were reported principally by Central Plains states, where most cases were presumably the result of spillover from rabid skunks. Eleven states reported > 10 cases of rabies in cats (Pennsylvania, 53 cases; Virginia, 34; New York, 23; Maryland, 21; North Carolina, 18; Texas, 15; Georgia, 15; Massachusetts, 15; New Jersey, 15; Kansas, 11; and Connecticut, 11). Twenty-one states did not report any rabid cats.

Dogs—Texas (15 cases), Puerto Rico (11), Georgia (6), and Kentucky (6) reported the largest numbers of cases of rabies in dogs (Figure 9). No other state reported > 5 cases of rabies in dogs in 2008. No cases were reported involving the dog/coyote rabies virus variant last identified in Texas in 2004. Twenty-eight states, the District of Columbia, and New York City did not report any rabid dogs.

Excluding rabid dogs from Puerto Rico, which are presumably infected with the mongoose rabies virus variant, 64 cases of rabies in dogs were reported from the United States. For 43 of these 64 (67%) cases, the variant was reportedly typed through the use of monoclonal antibodies or sequenced to determine the rabies virus variant. By comparison, the virus variant was typed in 58% of cases involving rabid dogs reported from the continental United States during 2007. One dog imported from Iraq to New Jersey was found to be infected with a canine rabies virus variant. Rabies virus variants isolated from all other rabid dogs that were typed in 2008 were reported as the terrestrial rabies virus variant associated with the geographic area where the dog was collected (Figure 1). Typing results were not reported from Arkansas (variant not typed for 3 of 4 rabid dogs), Georgia (2 of 6), Iowa (1 of 1), Kentucky (6 of 6), Massachusetts (1 of 1), North Dakota (4 of 4), New Jersey (1 of 2), and Pennsylvania (3 of 3).

Other domestic animals—The number of cases of rabies in cattle increased 3.5% from 57 in 2007 to 59 in 2008 (Figure 10; Table 1). Texas (9 cases), New York (6), Oklahoma (6), and Virginia (6) reported the largest numbers of rabid cattle. No other state reported > 5 cases of rabies in cattle in 2008. The 30 cases of rabies reported in horses and mules (including donkeys) in 2008 represented a 26.8% decrease from the 41 cases reported in 2007. Reported cases of rabies in sheep and goats decreased 7.7% from 13 cases in 2007 to 12 cases in 2008. A rabid llama was reported from Georgia.

Rabies in Humans

Two cases of rabies in humans were reported in the United States in 2008 (Table 2). In March 2008, a newly arrived immigrant from Mexico presented to a hospital in Santa Barbara, Calif, with encephalitic symptoms and died shortly thereafter. Rabies was suspected on the basis of the individual's clinical signs and reports of domestic and wild animal exposures acquired in Oaxaca,

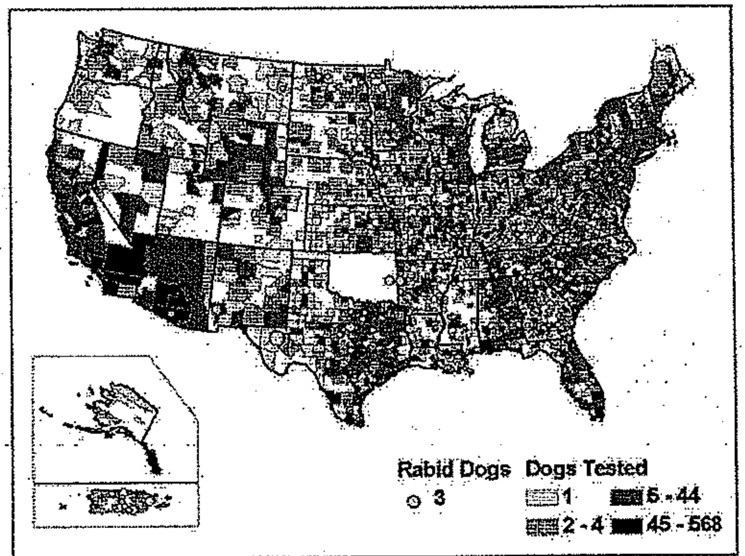

Figure 9—Reported cases of rabies in dogs, by county and municipio (Puerto Rico), 2008.

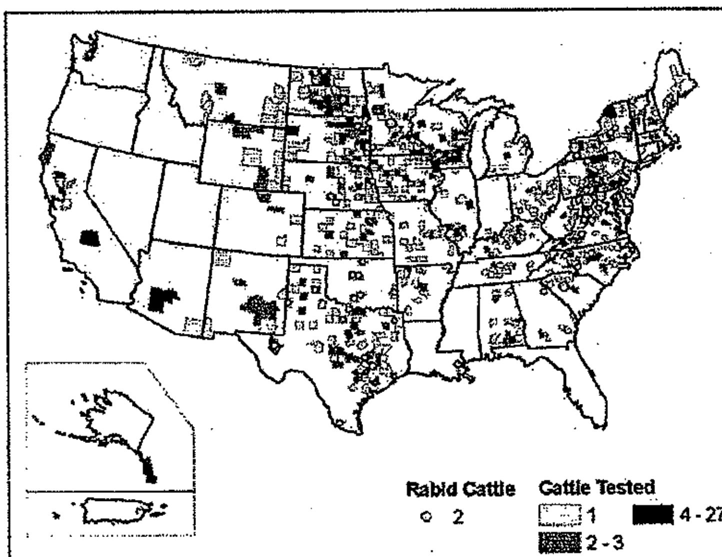

Figure 10—Reported cases of rabies in cattle, by county and municipio (Puerto Rico), 2008.

Table 2—Cases of rabies in humans in the United States and Puerto Rico, 2000 through 2008, by circumstances of exposure and rabies virus variant.

Date of death	State of residence	Exposure history*	Rabies virus variant†
20 Sep 00	CA	Unknown‡	Bat, Tb
9 Oct 00	NY	Bite-Ghana	Dog, Africa
10 Oct 00	GA	Unknown‡	Bat, Tb
25 Oct 00	MN	Bite	Bat, Ln/Ps
1 Nov 00	WI	Unknown‡	Bat, Ln/Ps
4 Feb 01	CA	Unknown‡-Philippines	Dog, Philippines
31 Mar 02	CA	Unknown‡	Bat, Tb
31 Aug 02	TN	Unknown‡	Bat, Ln/Ps
28 Sep 02	IA	Unknown‡	Bat, Ln/Ps
10 Mar 03	VA	Unknown‡	Raccoon, eastern United States
5 Jun 03	PR	Bite	Dog/mongoose, Puerto Rico
14 Sep 03	CA	Bite	Bat, Ln/Ps
15 Feb 04	FL	Bite	Dog, Haiti
3 May 04	AR	Bite (organ donor)	Bat, Tb
7 Jun 04	OK	Liver transplant recipient	Bat, Tb
9 Jun 04	TX	Kidney transplant recipient	Bat, Tb
10 Jun 04	TX	Arterial transplant recipient	Bat, Tb
21 Jun 04	TX	Kidney transplant recipient	Bat, Tb
Survived 04	WI	Bite	Bat, unknown
26 Oct 04	CA	Unknown‡	Dog, El Salvador
27 Sep 05	MS	Unknown‡	Bat, unknown
12 May 06	TX	Unknown‡	Bat, Tb
2 Nov 06	IN	Bite	Bat, Ln/Ps
14 Dec 06	CA	Bite	Dog, Philippines
20 Oct 07	MN	Bite	Bat, unknown
18 Mar 08	CA	Bite-Mexico	Bat, Tb related
30 Nov 08	MO	Bite	Bat, Ln/Ps

*Data for exposure history are reported only when the biting animal was available and tested positive for rabies, when plausible information was reported directly by the patient (if lucid or credible), or when a reliable account of an incident consistent with rabies exposure (eg, dog bite) was reported by an independent witness (usually a family member). †Variants of the rabies virus associated with terrestrial animals in the United States and Puerto Rico are identified with the names of the reservoir animal (eg, dog or raccoon), followed by the name of the most definitive geographic entity (usually the country) from which the variant has been identified. Variants of the rabies virus associated with bats are identified with the names of the species of bats in which they have been found to be circulating. Because information regarding the location of the exposure and the identity of the exposing animal is almost always retrospective and much information is frequently unavailable, the location of the exposure and the identity of the animal responsible for the infection are often limited to deduction. ‡In some instances in which the exposure history is unknown, there may have been known or inferred interaction that, especially for bats, could have involved an unrecognized bite.

Ln/Ps = *Lasionycteris noctivagans* or *Pipistrellus subflavus*, the silver-haired bat or the eastern pipistrelle. Tb = *Tadarida brasiliensis*, the Brazilian (Mexican) free-tailed bat.

Mexico. At autopsy, samples were submitted for rabies diagnosis, and results of a direct fluorescent antibody test performed by the California Department of Health Services were positive. Further testing by the CDC identified a novel rabies virus variant that phylogenetic analysis indicated was most closely related to rabies virus variants associated with free-tailed bats.[28]

On November 24, 2008, the CDC was contacted by the Missouri Department of Health and Senior Services regarding a potential case of human rabies. The patient was a 55-year-old male who had first presented to a hospital in southeastern Missouri on November 18 with chest and back pain. Clinical signs progressed to left arm paresthesia, dysphagia, erratic behavior, and hydrophobia. Rabies was suspected after the patient and family members related that approximately 4 to 6 weeks before the onset of clinical signs, the patient had been bitten on the ear by a bat. The patient had not sought rabies prophylaxis because the bat did not appear sick. On November 25, samples were submitted to the CDC and a diagnosis of rabies was confirmed. The virus was characterized as a rabies virus variant associated with silver-haired (*L noctivagans*) and eastern pipistrelle (*Pipistrellus subflavus*) bats. The patient died November 30 after 12 days of hospitalization.

During 2008, samples from 45 human patients in the United States were submitted to the CDC for rabies testing, representing a 24% decrease from the 59 samples tested during 2007. Primarily, prevention of human rabies consists of health communications to inform the public about proper behavior to decrease the likelihood of exposure, animal vaccination, and application of appropriate and timely postexposure prophylaxis in exposed humans.

Rabies in Canada and Mexico

Canada reported 235 laboratory-confirmed cases of rabies in domestic and wild animals in 2008. This was a decrease of 13.9% from the 273 cases reported in 2007 and was the seventh time in the past 8 years that there had been a decrease in the number of reported rabies cases. Eighty-seven percent (n = 204) of reported cases involved wild animals, 6.8% (16) involved livestock, and 6.4% (15) involved domestic companion animal species. There was also a decrease of 369 in the total number of diagnostic specimens tested in 2008, compared with 2007. The total number of diagnostic specimens tested has remained relatively stable over the 8-year period from 2001 through 2008 (mean, 7,962 samples tested/y). The number of rabid raccoons decreased by 54% (59 to 27), and rabid raccoons accounted for 11.5% of all rabid animals in 2008. Reported cases in bats and cattle decreased by 34% (93 to 61) and 20% (15 to 12), respectively. Increases occurred mainly in skunks, dogs, and foxes. Skunk cases increased by 27% (78 to 99), accounting for 42.1% of all rabies cases. Reported cases in dogs and foxes increased by 71% (7 to 12) and 15% (13 to 15), respectively. Some regional distribution of rabid wild species (eg, bats in British Columbia; skunks in Manitoba; foxes, skunks, and raccoons in Ontario; and raccoons in Quebec) was evident in Canada. No human cases of rabies were reported in Canada in 2008.

Mexico reported 232 cases of animal rabies in domestic and wild animals during 2008. This represented a 19.4% decrease from the number of cases (288) reported during 2007. Thirteen percent (31/232) of rabies cases involved dogs. Other domestic animals reported included cattle (183 [78.9% of all animals reported]) and other livestock (16 [6.9%]). Two cases of rabies were reported in wildlife species. Three cases of rabies in humans were reported during 2008; all were attributed to exposure to a vampire bat.

Discussion

Passive surveillance for rabies relies largely on interactions between humans and animal reservoirs and subsequent possible exposure of people to the rabies virus. Importantly, reporting of cases at an aggregate political boundary (ie, counties) complicates the ability to detect and analyze detailed relationships between environmental variables and the spread of zoonotic diseases such as rabies. Enhanced surveillance carried out by several state health departments and the USDA Wildlife Services augments passive public health surveillance in critical geographic areas, such as those areas ahead of epizootic fronts.

Although raccoons continued to account for the highest percentage (34.9%) of rabies cases reported among animals in the United States in 2008, the magnitude of this ratio has decreased consistently since 2004. Enzootic transmission of rabies among raccoons and from rabid raccoons to other species continued in 20 states, New York City, and the District of Columbia in 2008. The proportion of animal rabies cases geographically associated with the raccoon rabies virus variant reflected the high public health burden of this variant, compared with other terrestrial variants in the United States. Moreover, the human exposure risk to this variant is substantial, as reflected in cross-sectional studies of human postexposure prophylaxis.[29,30]

Rabid bats were reported from 47 of the 48 contiguous states during 2008. The epizootiology and phylogenetics of rabies in bats is distinct from the epizootiology and phylogenetics of terrestrial rabies maintained by mammalian carnivores. Knowledge regarding the circulation of rabies virus variants in bat species remains less developed than knowledge of variants found in carnivores. Bat-associated rabies virus variants account for most human infections in the United States in recent years. This trend has been highly publicized and resulted in public health recommendations for potential rabies exposures involving bats.[31,32] Increased publicity and awareness of bats and rabies have increased the rate of submission of bats for diagnostic testing following potential exposure. Since 1996, when the public health recommendations began to include more conservative guidelines regarding rabies exposure involving bats, the number of bats submitted for rabies testing has increased from approximately 10,000 to > 30,000 in 2008. Bats are submitted for testing at a rate that is comparable to that for cats and dogs, exceeding the submission rate of any other wildlife species.

Reports of rabid skunks increased in 2008. Given the results of antigenic typing of the virus from a subsample of rabid skunks from areas where raccoon rabies is enzootic, most rabid skunks in these states are presumed to be infected with the raccoon rabies virus variant. To date, studies have been unable to demonstrate evidence of unique adaptation, circulation, or maintenance of the raccoon rabies virus variant in skunks.[33] Approximately half of all reported skunks are infected with one of the skunk rabies virus variants. When skunks presumably infected with the raccoon rabies virus variant on the basis of geographic location were excluded, a dramatic decrease in the number of reported cases of rabies in skunks attributable to a skunk variant was observed.

In the southwest, Arizona reported an increase in the number of rabid skunks (n = 57). During 2001, a new focus of rabies in skunks related to a big brown bat rabies virus variant in the Flagstaff area of northern Arizona was recognized as having sustained transmission among skunks.[34,35] In response to this new variant, Arizona responded with trap, vaccinate, and release programs targeted at skunks as well as a field trial with a vaccinia–rabies glycoprotein recombinant vaccine to orally vaccinate skunks. Following 2 years of no reported cases involving this variant, a resurgence occurred during 2008. Responses similar to prior years are ongoing to try to control the spread of this novel variant.

The red fox rabies virus variant has not been detected in the northern United States in an excess of 5 years, most likely because of control measures (eg, oral vaccination programs) in place in Canada and the northern United States. Rabies in gray foxes in Arizona and Texas is typically the result of infection with gray fox variants found in each of those states. Oral vaccination of gray foxes in west and central Texas has been responsible for reducing the distribution of the Texas gray fox rabies virus variant. However, this oral vaccination program was challenged during 2008 as increased spillover into coyotes was observed, and cases were identified northwestward along the Pecos River outside the existing baiting zone. Additional baiting in these areas was initiated to provide extended coverage.

Throughout the western hemisphere, small mammals have never been implicated as potential reservoir species. Rabies among rodents and lagomorphs reflects spillover infection from regional terrestrial reservoir species. Among rodents, rabies occurs primarily in groundhogs (31 cases reported in 2008) in areas of the country affected by the raccoon rabies virus variant.[36] Rabies is occasionally reported in other large-bodied members of this order, such as beavers (1 case in 2008). Large-bodied wild rodents and captive rabbits in outdoor cages or pens may become infected and survive long enough to pose a risk to other species, such as humans.[37] Rabies is seldom reported in smaller rodents, presumably because of the high likelihood of death or severe trauma in small rodents attacked by rabid carnivores. There has been no documentation of rabies virus transmission from a rodent or lagomorph to a human.

Despite the threat of rabies transmission from wild terrestrial carnivores, the use of population-reduction programs to control rabies among such animals is not desirable. Use of an oral vaccination program in Switzerland during the past 30 years resulted in a declaration of rabies-free status for that country in 1998, and similar strategies led to rabies-free status being declared in France in 2000 and in Germany in 2008.[38,39] The elimination of a rabies virus variant associated with red foxes in southern Ontario also supports the hypothesis that rabies virus variants associated with foxes can be eliminated through oral vaccination programs.[40]

In the United States, oral rabies vaccination programs may have restricted the expansion of raccoon rabies. Programs involving distribution of the vaccinia–rabies glycoprotein recombinant vaccine in baits to prevent or slow the geographic expansion of rabies in wild raccoons continue in a number of states and are being expanded. During 2008, multiple state agencies, USDA Wildlife Services, and the CDC continued to cooperate in a massive undertaking to maintain and expand an "immune barrier" beginning in Ohio, Pennsylvania, and New York and intended to reach the Gulf of Mexico in Alabama in an attempt to curtail the spread of raccoon rabies. In Ohio, Pennsylvania, Maryland, West Virginia, Virginia, North Carolina, and northeastern Tennessee (otherwise known as the Appalachian Ridge oral rabies vaccination zone), approximately 5 million doses of vaccinia–rabies glycoprotein recombinant vaccine–laden baits were distributed. In addition, approximately 1 million doses of oral vaccine were distributed in Georgia, Alabama, and Tennessee (the GAT oral rabies vaccination zone).[41] Approximately 3 million baits were distributed in Texas in an attempt to contain and eliminate the gray fox rabies virus variant and prevent the reintroduction of canine rabies virus variants associated with coyotes and dogs from Mexico during 2008.[8–10] Enhanced surveillance conducted by USDA Wildlife Services and routine surveillance by state public health agencies continue to determine the placement of new oral rabies vaccination zones as well as the shape of baiting zones each year. Translocation of infected animals, as has occurred in the past, continues to pose a substantial threat to the goals of national oral rabies vaccination programs.[5,42] Concerns regarding vaccine safety and efficacy, ecologic impact, and physical bait variables, which were raised during earlier trials, continue to be assessed.[17,18,42–46] Development of novel biologics is ongoing to overcome the limited efficacy of the vaccinia–rabies glycoprotein recombinant vaccine in certain animal species (eg, skunks and mongooses).[47–50]

Despite little change in the total number of overall reported rabies cases in domestic animals, a 19.4% decrease in the reported cases of rabies in dogs occurred from 2007 to 2008. Cases of rabies in cats and dogs are primarily attributable to spillover from local terrestrial reservoirs,[51] and the United States has been free from dog-to-dog transmission of rabies since 2004.[52,53] However, continued surveillance will be required for early detection and to prevent this rabies virus variant or others from being reintroduced into the United States. The potential for reintroduction has been demonstrated in recent years. Following the importation of a rabid puppy from India in 2007, a rabid dog was imported from Iraq into New Jersey during 2008.[54] Since 2004, at least 4 cases of rabies in recently imported dogs have been reported. The risk of importation of rabies and other exotic zoonotic diseases may be increasing as more animals are brought into the United States each year.[55]

Since 1992, cats have remained the leading domestic animal species with rabies reported each year.[56] Several studies[29,30] have indicated that cats are a leading domestic animal source of possible human exposure to rabies requiring postexposure prophylaxis. Further reduction in the number of rabies cases in companion species, especially cats, may require stricter observance and enforcement of vaccination and supervision. Vaccination remains a crucial element in this effort. However, not all states currently have laws requiring vaccination of cats against rabies.

Rabies vaccination of pet mammals and livestock that have regular contact with people is a fundamental barrier to human exposure. A single incident involving a case of rabies in a companion animal species can result in large economic expenditures and public health efforts to ensure that human disease does not occur.[57–59] Although widespread vaccination of livestock is neither economically feasible nor justifiable on public health grounds, vaccination of valuable livestock or livestock that may have regular contact with human beings (eg, animals in petting zoos) in rabies epizootic areas should be considered.[60,61]

Twenty-eight cases of human rabies have been reported in the United States since 1998, including the 2 cases reported in 2008. Seven of these 28 (25%) individuals were infected outside the continental United States (6 abroad and 1 in Puerto Rico). Most human rabies infections that occur in foreign countries where dog rabies is enzootic involve regional canine rabies virus variants, with the exception of the 2008 case from Mexico, which was associated with wildlife species. Twenty-one of the 28 (75%) individuals were infected with rabies virus variants indigenous to the United States. Analysis of monoclonal antibodies and genetic sequencing data indicated that 17 of these 21 (81%) persons were infected with bat rabies virus variants. Epidemiologic investigations in 3 additional cases implicated a bat as the most likely source of exposure. In 1 case, the virus was typed as a rabies virus variant associated with raccoons. In 13 of the 20 (65%) human rabies cases associated with bats since 1998, there has been a report of a bite or direct contact with a bat (eg, awaking to find a bat on the body or picking up a grounded bat). Four (20%) cases were associated with organ transplants or an arterial graft from a rabies-infected donor.[4,22] Three patients with bat-associated rabies were reported to have no known exposure to a bat. In these instances, the most likely route of infection with rabies virus was a bite that was ignored or went unnoticed during an interaction with a bat. Although rabies infection of humans following exposure to bats remains a rare occurrence, the prevention of such infections remains an important public health concern.

Rabies should be included in the differential diagnosis for any patient with unexplained, acute, rapidly progressive encephalitis, especially in the presence of autonomic instability, dysphagia, hydrophobia, paresis, or parasthesia.[62] Given the report[63] of survival of a rabies patient after experimental treatment in 2004, early diagnosis of potential rabies cases has become increasingly important, particularly if experimental treatment is to be considered. However, the benefits of any particular experimental rabies treatment regimen have not been determined. No single course of treatment for rabies in humans has been documented to be efficacious after clinical signs of rabies are present.

New Advisory Committee on Immunization Practices recommendations on human rabies prevention were issued in May 2008, in addition to an update of the national rabies compendium.[31,60] The committee's recommendations were updated to provide an evidence-based approach to current recommendations for rabies preexposure vaccination and postexposure prophylaxis, providing a review of current knowledge on human rabies vaccines, rabies immune globulin, prophylaxis series, and adverse events. Overall recommendations regarding postexposure prophylaxis did not change from the 1999 recommendations.

During 2008, ongoing rabies vaccine supply issues reinforced the need to emphasize basic human rabies prevention and prophylaxis recommendations as well as animal rabies control. Circumstances initiated in late 2007 led to limitations in the available supply of both of the commercially licensed human vaccines in the United States. A national working group of subject matter experts, consisting of state and federal health officials, experts from academia, and representatives from relevant professional organizations, was convened to provide guidance and recommendations in response to the limited rabies vaccine supply and in the event of a true shortage (defined as the point at which vaccine would not be projected to be available for persons with rabies exposure). Throughout most of 2008, rabies vaccine was restricted to use for postexposure prophylaxis only, except for use in critical first responders (eg, rabies diagnosticians) by approval of state and federal public health officials. To prevent a shortage, close consultation with local and state health departments was recommended before initiation of postexposure prophylaxis after a potential exposure. This extensive public health response prevented a true shortage of rabies vaccine throughout the summer during peak rabies season, and supplies improved toward the end of 2008 and into 2009. The national working group continues to evaluate recommendations for rabies postexposure prophylaxis in the event of a shortage, improvements to human rabies postexposure prophylaxis surveillance, and national stockpile options.

2009 Rabies Update

A preliminary analysis of data from states submitting monthly data to the CDC for the first 4 months of 2009 showed a decrease in the number of cases of rabies, compared with the same time period during 2008. One case of human rabies was reported from Texas during the first 5 months of 2009. In March 2009, a 17-year-old female presented to a hospital in Houston with a history of headaches, photophobia, and left-sided weakness. The condition worsened over the next several days, and the patient was hospitalized with acute neurologic abnormalities and aggressiveness. Results of all routine tests for suspected conditions were negative. The patient had no history of foreign travel. During follow-up, the patient reported a visit to a Texas cave where she had had direct contact with bats but no reported bite. Samples were submitted to the CDC to rule out rabies. Antibodies to rabies virus were detected in the patient's CSF, but no viral amplicons were detected in saliva or in a nuchal biopsy specimen. The patient's condition improved gradually, and she was discharged without incident.

Rabies vaccine supplies showed improvement over the limitation observed in 2008. One vaccine, RabAvert, remained available for both pre- and postexposure prophylaxis, whereas the vaccine Imovax was available only for postexposure prophylaxis following consultation with a state health department. Published evidence regarding the necessity of the fifth dose of vaccine in patients undergoing rabies postexposure prophylaxis was prepared for review by the Advisory Committee on Immunization Practices. At its June 24, 2009, meeting, the committee's rabies working group presented evidence in support of a recommendation to reduce the number of vaccine doses in the human rabies postexposure prophylaxis series from 5 to 4 doses. After much discussion, the committee voted in favor of accepting the recommendation. This will effectively change the 2008 Advisory Committee on Immunization Practices' recommendations for human rabies prevention as follows: the postexposure prophylaxis protocol will consist of administration of human rabies immune globulin (20 U/kg) on day 0 and administration of 4 doses of vaccine (1 mL, IM) on days 0, 3, 7, and 14. Formal publication of the recommendations will be forthcoming.

a. SIEPI Epidemiological Information System [database online]. Washington, DC: Pan American Health Organization, Pan American Center for Foot-and-Mouth Disease, 2008. Available at: siepi.panaftosa.org.br/Export.aspx. Accessed Jul 15, 2009.

b. ArcMap, version 8.3, ESRI, Redlands, Calif.

Parvovirus Outbreak in Raccoons (*Procyon lotor*) being Rehabilitated at Wildcat Wildlife Center

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Abstract: Parvovirus infection in raccoons (*Procyon lotor*) is a potentially devastating disease for rehabilitation centers that work with large numbers of raccoons annually. Raccoons are reportedly affected by three related parvoviruses including raccoon parvovirus, feline parvovirus (panleukopenia or feline distemper), and mink parvovirus (mink enteritis virus). The disease primarily affects young raccoons and is characterized by depression, bloody diarrhea, and sudden death. The importance of parvoviral infections in free-ranging raccoons is not known but is potentially a substantial source of mortality. During July and August of 2007, a parvovirus outbreak killed 26 of 98 raccoons being rehabilitated at the Wildcat Wildlife Center (WWC) in Delphi, Indiana. The raccoons presented with lethargy, anorexia, diarrhea, and loss of motor skills. Euthanasia was necessary for several of the severely affected animals. Two animals were submitted to the Animal Disease Diagnostic Laboratory in West Lafayette, Indiana, where veterinary pathologists reported myeloid hypoplasia and histopathologic lesions consistent with parvoviral enteritis, although no parvoviral particles were detected with virus isolation. Acutely affected raccoons found alive were isolated and given antibiotics and supportive care with little success. One recovered and likely will be suitable for release. The other 71 raccoons were successfully released. At the time of the outbreak, all but three of the rehabilitating raccoons were fully vaccinated for recommended canine and feline diseases, including parvovirus.

Keywords: Raccoon, *Procyon lotor*, parvovirus, wildlife, vaccination

INTRODUCTION

Raccoons are susceptible to three antigenically similar but unique parvoviruses including raccoon parvovirus, panleukopenia (feline parvovirus, feline distemper), and mink parvovirus (Barker et al 1983; Doster 2006; Shenoy 2007). They are not affected by canine parvovirus (Barker et al 1983; Raymond 1997). Parvovirus tends to have the greatest effect on young or immunocompromised animals (Martin and Zeidner 1992; Shenoy 2007). Affected raccoons present with weakness, diarrhea, dehydration, and depression with rapid deterioration (Davidson 1997). They may have a loss of fear of humans (Raymond 1997). The importance of parvoviral infections in free-ranging raccoons is not known, but is potentially a substantial source of mortality (Davidson 1997). There have been reports of severe disease in free-ranging raccoons with parvovirus infection, but the presence of concurrent infections such as cryptosporidiosis and coronavirus makes it difficult for a definitive conclusion to be made about the role each agent plays in disease (Martin and Zeidner 1992). In captive settings such as rehabilitation facilities, where large numbers of young raccoons are housed together or in close proximity, parvovirus infection may be a more serious threat. The Southeastern Cooperative Wildlife Disease Study reported that since 1976, 12 out of 655 examined raccoons tested positive for parvovirus and 92 percent of those had a history of captivity (Doster 2006). Although the history of these raccoons and why they were tested is not completely clear, it appears that time in captivity is a risk factor for parvovirus in raccoons.

SUMMARY OF PARVOVIRUS OUTBREAK

Between 11 July and 12 August 2007, a parvovirus outbreak killed 26 raccoons being rehabilitated at WWC in Indiana. A total of 98 raccoons ranging from three to six months of age were being housed at the time of the outbreak. There are four separate raccoon areas at WWC where animals are housed, with groups of two to seven housed in wire pens and larger groups (7 to 21) in two corn cribs modified for raccoon caging. Direct contact between the rehabilitating raccoons and other free roaming wildlife such as raccoons and mink, as well as domestic feral cats is possible through the fencing. At the time of the outbreak,

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all but three of the rehabilitating raccoons were fully vaccinated for recommended canine and feline diseases including parvovirus. Vaccinations begin when raccoons are about three weeks old with the Duramune Max 5-CvK® (Fort Dodge, Overland Park, KS) vaccine without leptospira followed by a Duramune 5-CvK/4L® (Fort Dodge, Overland Park, KS) booster every three weeks for at least three sets. These vaccines are used to protect against canine distemper and leptospirosis. Fel-O-Vax IV® (Fort Dodge, Overland Park, KS) plus Calicivax® vaccines (Fort Dodge, Overland Park, KS) are given on alternate weeks relative to the canine vaccine. This vaccine is used to protect against feline and raccoon parvovirus. Due to difficulty finding a supplier and shipping delays, a mink parvovirus enteritis vaccine was not available until after this outbreak had largely subsided. The Biovac vaccine by United Vaccines Co. (Madison, WI) is now included in raccoon vaccination protocols at WWC.

Symptoms and Treatment. The 27 clinically affected raccoons presented with lethargy, anorexia, green to olive diarrhea, oculonasal discharge, loss of motor skills, or sudden death. Two raccoons exhibited black, tarry stool approximately 24 hours before they died. Sick raccoons were immediately isolated indoors and given amoxicillin/clavulanic acid (Clavamox®, Pfizer Animal Health, New York, NY), sulfadimethoxine (Albon®, Pfizer, New York, NY) and subcutaneous fluids (Normosol-R®, Johnson & Johnson Medical, Division of Ethicon, Inc., Arlington, TX). One animal recovered, but the other 26 continued to deteriorate and later died. Euthanasia was necessary for six severely affected animals. More drastic measures, such as continuous intravenous fluid support, likely would have been more effective but were not practical. There are no reports in the literature describing effective treatment of advanced parvoviral enteritis in raccoons. In kittens, intensive parenteral fluid and antibiotic therapies reduce mortality (Green and Addie 2006).

Two of the euthanized raccoons were submitted to the Animal Disease Diagnostic Laboratory in West Lafayette, Indiana where veterinary pathologists reported myeloid hypoplasia and histopathologic lesions consistent with parvoviral enteritis, although no parvoviral particles were detected with virus isolation. Other findings included intestinal coccidiosis and positive *Salmonella* sp. culture. The significance of concurrent coccidiosis and salmonellosis in the examined raccoons is unclear. Both are reported in raccoons and both may have contributed to the clinical disease and death of the animals. Neither was likely the primary cause of death. Coccidiosis is more commonly a subclinical finding but severe disease can occur secondarily in immunocomprimised domestic carnivores (Dubey and Greene 2006). Salmonellosis is usually associated with mild to moderate gastroenteritis but can cause severe septicemias secondary to immunosuppression (Ketz-Riley 2003). The raccoons may have become infected secondarily after being immunocompromised by parvovirus. Concurrent infections seem to be a frequent finding in wildlife with clinical disease. Martin and Zeidner (1992) reported a similar situation in which a raccoon diagnosed with parvoviral enteritis had concurrent cryptosporidosis and coronavirus infection.

Biosecurity Measures. After the outbreak began, WWC staff immediately placed bleach water footbaths at entrances to the hospital, isolation rooms, and other key locations. Footbaths were changed at least twice per day. Volunteers were assigned different raccoon areas and instructed to avoid walking between areas. After completing their area, volunteers either showered and changed clothes, or left the facility. Anyone who worked with the raccoons was not allowed to care for other mammals that day. All tools, including buckets, nest boxes, enrichment items, wheelbarrows, pool liners, and food dishes were dedicated to one area and scrubbed with soapy bleach water, rinsed, and dried after each use. Parvovirus is hardy in the environment but can be killed by certain disinfectants including sodium hypochlorite (bleach) and glutaraldehyde (Gaskell and Dawson 2005). Virkon S® (Global International Inc., St. Louis, MO) and Trifectant® (Vétoquinol USA Inc., Buena, NJ) disinfectants are also good choices for parvovirus; the primary agent in these disinfectants is potassium peroxymonosulfate.

CONCLUSION

The outbreak at WWC raises major concerns regarding the management of parvovirus and other infectious disease outbreaks in the wildlife rehabilitation setting. It is obvious that once clinical disease becomes apparent, it is almost always too late to manage the outbreak medically without significant mortalities. This is especially true in the wildlife rehabilitation setting where the majority of patients are weak orphans or trauma patients that simply cannot handle any additional stress or illness. Preventative measures might include double fenced outdoor enclosures, and sanitation/disinfection of pens, water bowls, enrichment items, and other objects that may act as fomites. Young or healthy animals should always be cared for before older or sick animals to reduce the spread of disease to susceptible animals. Ideally, these measures should be ongoing at all times. The feasibility and practicality of many of these measures, however, is often questionable in the rehabilitation environment due to lack of money, space, or staff. In addition, outdoor enclosures on natural substrates are notoriously difficult to disinfect. Careful attention to these easily ignored matters can significantly reduce the impact of an infectious disease outbreak. The measures taken by WWC early in the course of the outbreak likely limited the spread of parvovirus and minimized mortality to 26 of 98 at risk animals.

This outbreak represents the first significant raccoon loss since vaccination protocols were initiated at WWC and raises major concerns about vaccine efficacy. The efficacy of vaccines designed for domestic animals and used in wildlife has not been well studied. Protocols are often based on clinical experience or by extrapolating data from studies on domestic animals (Shenoy 2007). This can make interpretation of the efficacy of vaccine protocols in the wildlife rehabilitation setting difficult to determine or improve. In this case, all but three of the affected raccoons were fully vaccinated with recommended parvovirus vaccines. There are many possible reasons why the vaccinated raccoons may not have been adequately protected. They may have been too stressed from recent weaning and moving from indoors to outdoors to mount an adequate immune response to the vaccine antigens. Dog and cat vaccines may not provide adequate cross protection against raccoon or mink parvovirus, or there simply could have been a bad batch of vaccine or an undetected problem with vaccine storage. It may be that the sick raccoons had not had sufficient time to mount a suitable response prior to exposure to parvovirus in the environment. This occurs when inactivated vaccines are used in exposed kittens (Greene and Addie 2006). Early and prolonged vaccination schedules are recommended for at risk kittens (Greene and Addie 2006). Additional research and collaboration between rehabilitators and wildlife veterinarians is needed. At WWC, it will be important to continue monitoring outbreaks and their severity over the next few years to determine the source of the parvovirus, evaluate vaccine efficacy, implement practical preventative measures, and expose possible internal oversights that may be spreading virus between pens.

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Baylisascaris procyonis: An Emerging Helminthic Zoonosis

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▸ See letter "Thermal Death Point of *Baylisascaris procyonis* Eggs" in volume 13 on page 172.

▸ This article has been cited by other articles in PMC.

Abstract

Baylisascaris procyonis, a roundworm infection of raccoons, is emerging as an important helminthic zoonosis, principally affecting young children. Raccoons have increasingly become peridomestic animals living in close proximity to human residences. When *B. procyonis* eggs are ingested by a host other than a raccoon, migration of larvae through tissue, termed larval migrans, ensues. This larval infection can invade the brain and eye, causing severe disease and death. The prevalence of *B. procyonis* infection in raccoons is often high, and infected animals can shed enormous numbers of eggs in their feces. These eggs can survive in the environment for extended periods of time, and the infectious dose of *B. procyonis* is relatively low. Therefore, the risk for human exposure and infection may be greater than is currently recognized.

Key words: *Baylisascaris procyonis*, larva migrans, epidemiology.

Baylisascaris procyonis, a ubiquitous roundworm infection of raccoons (*Procyon lotor*), is increasingly being recognized as a cause of severe human disease (1,2). *B. procyonis* has a widespread geographic distribution, with infection rates as high as 70% in adult raccoons and exceeding 90% in juvenile raccoons (3). As with other ascarids, eggs are excreted in feces and must develop externally, typically in soil, to become infectious. When raccoons ingest infective eggs, larvae will hatch, enter the wall of the small intestine, and subsequently develop to adult worms in the small bowel. However, ingestion of eggs by other host animals, especially rodents and other small mammals, results in extraintestinal migration of larvae (4); an estimated 5%-7% of larvae invade the brain (5). The migration of helminth larvae through tissue in suboptimal hosts is termed larva migrans and may affect the viscera (visceral larva migrans [VLM]), the eye (ocular larva migrans [OLM]), or the nervous system (neural larva migrans [NLM] (6). Raccoons may also become infected when they eat larvae that have become encapsulated in the tissues of rodents and other animals (3).

More than 90 species of wild and domesticated animals have been identified as infected with *B. procyonis* larvae (3). Outbreaks of fatal central nervous system disease caused by *B. procyonis* have occurred on farms and in zoos and research animal colonies and have affected commercial chickens, bobwhite quail, guinea pigs, commercial pheasants, and domestic rabbits (7–11). Natural infections have also been recognized in dogs, rodents, porcupines, chinchillas, prairie dogs, primates, woodchucks, emus, foxes, and weasels (12–16). Experimental infection of a variety of nonhuman primates has also been reported (17).

Human Infection

B. procyonis infection of humans typically results in fatal disease or severe sequelae (1,2,18-24; pers. comm., W. Murray). Clinical manifestations include eosinophilic encephalitis, ocular disease, and esoinophilic cardiac pseudotumor. Elevated peripheral cerebrospinal fluid eosinophilia can be detected in cases of meningoencephalitis. Eleven recognized human cases, four of them fatal, have been reported (Table). The first human case was reported in 1984 in a 10-month-old infant with fatal eosinophilic meningoencephalitis (18). At autopsy, numerous granulomas containing larvae of *B. procyonis* were observed in several organs and tissues (18). The brain was the most heavily affected, with granulomas concentrated in the periventricular white matter, around the dentate nuclei, and along the cerebral and cerebellar cortices. Numerous granulomas and larvae were also found in the mesentery and cardiac tissue. The infant's family lived in a rural, wooded area of Pennsylvania, and raccoons were nesting in unused chimneys at the time infection was acquired.

Table
Reported human cases of larval *Baylisascaris procyonis* infection

Four additional cases of eosinophilic encephalitis with similar pathologic characteristics have been documented. Magnetic resonance images from a human case of *Baylisascaris* encephalitis are shown in Figure 1. In patients who have survived central nervous system (CNS) invasion, severe neurologic sequelae have resulted. In a fatal case, an eosinophilic cardiac pseudotumor, affecting principally the left ventricle, was observed at autopsy; no larvae or granulomas were found in any other tissue examined.

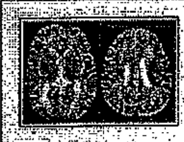

Figure 1
Biopsy-proven *Baylisascaris procyonis* encephalitis in a 13-month-old boy. Axial T2-weighted magnetic resonance images obtained 12 days after symptom onset show abnormal high signal throughout most of the central white matter (arrows) compared with the (more ...)

No effective therapy exists for the visceral form of *B. procyonis* larval infection. In an experimental model, mice treated with albendazole and diethylcarbamezine within 10 days after infection were protected from CNS disease (25); however, several anthelminthic agents have been used to treat human cases without success. Laser photocoagulation has been successful in treating ocular infection

(26).

Because the disease is transmitted by the fecal-oral route, human cases of *B. procyonis* infection typically occur in younger age groups, mainly infants, who often engage in oral exploration of their environment and are therefore more likely to be exposed to *B. procyonis* eggs. Raccoon activity near the patient's residence is often described. All but one of the reported patients to date have been male; however, there is no reason to believe that females are less susceptible to infection.

Diagnosis and Underrecognition of Infection

Diagnosis of *B. procyonis* infection is typically done through morphologic identification of larvae in tissue sections (27). However, accurate diagnosis requires experience in recognizing larval morphologic characteristics and differentiating among a number of possible larval nematode agents, including *Toxocara canis*, *T. cati*, *Ascaris lumbricoides*, and species of *Gnathastoma*, *Angiostrongylus*, and *Ancylostoma*, as well as larval cestode infections such as cysticercosis and echinococcosis (6,27). Characteristic features of *B. procyonis* larvae in tissue include its relatively large size (60 µ) and prominent single lateral alae (27) (Figure 2). While serologic testing has been performed in some cases as supportive diagnostic evidence, no commercial serologic test is currently available (28,29). However, a presumptive diagnosis can be made on the basis of clinical (meningoencephalitis, diffuse unilateral subacute neuroretinitis [DUSN], pseudotumor), epidemiologic (raccoon exposure), radiologic (white matter disease), and laboratory results (blood and CNS eosinophilia).

Figure 2
Cross-section of *Baylisascaris procyonis* larva in tissue section of brain, demonstrating characteristic diagnostic features including prominent lateral alae and excretory columns.

Human baylisascariasis is probably underrecognized, and the full spectrum of clinical illness is unclear. The agent is unknown to most clinicians and typically is not considered in a differential diagnosis. In addition, confirming the diagnosis requires an effective biopsy specimen that must contain an adequate cross-section of a larva. Since small numbers of larvae can cause severe disease and larvae occur sporadically in tissue, a biopsy may frequently fail to include larvae; such a specimen will result in a negative finding. Moreover, larval morphologic characteristics may not be recognized or may be misidentified. The accurate diagnosis of parasites in tissues can be difficult even for trained microscopists, and mistaken identification, particularly of helminth larvae, is not uncommon (27). Finally, no commercial serologic test exists for the diagnosis of *B. procyonis* infection, and the sensitivity, specificity, and predictive value of available serologic tests are unknown. Evidence for underrecognition of larval *B. procyonis* infection can be found in several reported cases of DUSN caused by larvae compatible with *B. procyonis* and a case of eosinophilic meningoencephalitis reported in an infant in 1975 (26,30,31).

Infection Potential and Human Risk

Although relatively few human cases of baylisascariasis have been reported, several factors suggest that the likelihood of exposure and infection may be greater than is currently recognized. Raccoons have a widespread geographic distribution, and infection with *B. procyonis* is common in raccoon populations, with typically high prevalence rates observed. An infected raccoon can harbor numerous adult worms and may excrete large numbers of eggs. A single adult female worm may produce an estimated 115,000 to 877,000 eggs per day, and an infected raccoon can shed as many as 45,000,000 eggs daily (3,4,32). In light of the relatively low infectious dose of *B. procyonis* (estimated to be ≤5,000 eggs) and the viability of the eggs in the environment for months to years, the infection potential is not insubstantial. Raccoons have increasingly become peridomestic animals living in close proximity to human residences and are among the fastest growing wildlife populations nationwide. These animals benefit from feeding on abundant pet food left accessible, either accidentally or intentionally, and their populations can thrive under such conditions. In one suburban area near the residence of a recent patient in northern California, the raccoon population was measured at 30 animals per quarter acre. Areas frequented by raccoons and used for defecation were found in close proximity to human dwellings, and *B. procyonis* eggs are routinely recovered from these areas (1). Children, particularly toddlers, may be at particular risk of exposure.

Although baylisascariasis may indeed be underdiagnosed, asymptomatic human infection may be the typical response, and the limited number of cases reported may indicate that an unrecognized immune defect is necessary for severe infection to occur. The prevalence of asymptomatic infection in human populations has yet to be determined.

A Possible Agent of Bioterrorism

In an era of increasing concern about bioterrorism (33), certain characteristics of *B. procyonis* make it a feasible bioterrorist agent. The organism is ubiquitous in raccoon populations and therefore easy to acquire. Enormous numbers of eggs can be readily obtained, and these eggs can survive in an infectious form for prolonged periods of time. As with other ascarids, the eggs can remain viable in a dilute (0.5%–2%) formalin solution for an indefinite period of time, and animal studies suggest that *B. procyonis* has a relatively small infectious dose. Moreover, the organism causes a severe, frequently fatal infection in humans, and no effective therapy or vaccine exists. Introduction of sufficient quantities of *B. procyonis* eggs into a water system or selected food products could potentially result in outbreaks of the infection. A similar agent, *Ascaris suum*, a roundworm of pigs, was used to intentionally infect four university students who required hospitalization after eating a meal that had been deliberately contaminated with a massive dose of eggs (34). Contamination of community water sources would be difficult since the eggs of *B. procyonis* are relatively large (80 µm long by 65 µm wide) and would be readily removed by standard filtration methods or the flocculation and sedimentation techniques used by municipal water systems in the United States. However, posttreatment contamination or targeting of smaller systems could be possible.

Conclusion

Baylisascariasis is an emerging helminthic zoonosis with the potential for severe infection that may be a more important public health problem than is currently recognized. Educating the medical community is of paramount importance in helping to define the extent of infection. Physicians should consider *B. procyonis* infection in the differential diagnosis of patients with eosinophilic meningoencephalitis, DUSN, and eosinophilic pseudotumor. While infants and children have a higher probability of infection, all age groups are at risk. The public should be made aware of the potential risks of exposure to raccoons and raccoon feces. Raccoons should be discouraged as pets or should be routinely evaluated for *B. procyonis* infection and treated. However, screening and treatment may not be sufficient to prevent exposure, since the likelihood of reinfection is high. The public should be discouraged from feeding raccoons and should ensure that possible food sources (such as pet food, water, and garbage) are protected from raccoon access. Further study of the impact of larval *B. procyonis* infection on human health is warranted. Development of a standardized serologic test for *B. procyonis* would allow epidemiologic studies of its prevalence and incidence and help determine factors associated with infection. A sensitive and specific test would also provide a noninvasive method of diagnosis. Finally, a better understanding of the pathogenesis of *B. procyonis* infection and efforts to develop effective treatment approaches are warranted.

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The authors thank Howard A. Rowley for graciously providing the images in Figure 1.

Biography

- Dr. Sorvillo is Associate Professor, Department of Epidemiology, UCLA School of Public Health. His research interests include the epidemiology and control of infectious diseases, particularly parasitic agents.

Footnotes

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SURVIVAL AND MOVEMENTS OF TRANSLOCATED RACCOONS IN NORTHCENTRAL ILLINOIS

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Abstract: Translocation of nuisance raccoons (*Procyon lotor*) is a common practice, but the fates of translocated animals after release are not known. We monitored postrelease survival rates and dispersal of radiocollared raccoons that were trapped as nuisance wildlife in suburban Chicago and translocated to a rural forest preserve (translocated urban), trapped in another wooded area and translocated to the forest preserve (translocated rural), and trapped and released in the forest preserve that served as the release site for the translocations (resident). Thirty-one raccoons were radiotracked in autumn 1993, and 45 were radiotracked in autumn 1994. We detected no differences in survival rates among the 3 treatment groups ($P > 0.05$). Resident raccoons tended to remain in the vicinity of the release site, whereas translocated raccoons left the release site within hours to days and dispersed into the surrounding area. Dispersing raccoons had high daily movement rates for the first 2 weeks postrelease but then seemed to establish new home ranges. Translocated raccoons frequently denned near human residences and in agricultural fields, whereas resident raccoons denned primarily in the forest preserve. Because translocated raccoons survived well, translocation could be an effective way to supplement depleted or reestablish extirpated populations of this species. However, translocating large numbers of raccoons for animal damage control could cause problems for other wildlife and human residents near release sites, and translocated animals could serve as vectors for wildlife diseases during zoonotic outbreaks.

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Key words: animal damage control, Illinois, nuisance wildlife, *Procyon lotor*, raccoon, survival rates, translocation.

Many species of wildlife are thriving in human-dominated landscapes. In the midwestern United States, for example, some species of game and furbearing mammals such as white-tailed deer (*Odocoileus virginianus*), coyotes (*Canis latrans*), raccoons (*Procyon lotor*), and opossums (*Didelphis virginiana*) are probably near historical record high numbers (Hoffmeister 1989). As populations of humans also continue to increase, conflicts between humans and wildlife are inevitable. In 1994, for example, 45,331 mammals were handled by nuisance wildlife control permittees in Illinois (Bluett 1995). Raccoons accounted for 13,193 (29%) of these mammals, and were by far the most common pest species. Illinois law allows for nuisance raccoons to be euthanized, but many are relocated to woodlots or forest preserves in rural areas. A total of 18,879 mammals was reported as translocated and released in Illinois in 1994, including 5,832 raccoons (Bluett 1995). These numbers are typical of recent years, but they probably underestimate the true numbers of translocated animals because many residents of rural areas handle nuisance wildlife problems without reporting them to state agencies.

Translocating nuisance wildlife to rural habitats might seem a humane way to handle problem animals. However, survival rates of translocated animals after release are not known. Translocated deer, for example, generally have high mortality rates (O'Bryan and McCullough 1985, Jones and Witham 1990, Bryant and Ishmael 1991). Radiotelemetry studies of translocated raccoons have yielded mixed results. Frampton and Webb (1973) and Taylor and Pelton (1979) reported high survival rates among translocated raccoons, but Wright (1977) and Rosatte and MacInnes (1989) found more than half of the raccoons translocated in their studies died within a few months of release. Few studies have monitored the fates of raccoons translocated from urban to rural environments (Rosatte and MacInnes 1989), and no published studies have simultaneously monitored survival rates of resident raccoons at the release site.

Translocating wildlife could facilitate the spread of zoonotic diseases (Davidson and Nettles 1992). For example, the rapid spread of rabies in raccoons in the eastern United States

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has been linked to translocation of disease-carrying animals (Nettles et al. 1979, Smith et al. 1984, Jenkins and Winkler 1987). If translocated raccoons settle near human residences, they could add to nuisance wildlife problems in rural areas. Finally, translocated raccoons could compete with resident populations for food or other resources, disrupt existing social structure of resident populations, or increase rates of predation on songbird and waterfowl nests or other threatened wildlife.

In this study, we compared survival rates, dispersal from the release area, and causes of mortality among raccoons translocated from an urban to a rural area, translocated from a rural to a rural area, and, as a control, residents captured at the rural release site. We recorded habitats used as daily den sites by translocated and resident raccoons as an indicator of whether translocated raccoons were likely to add to wildlife nuisance problems for human residents of rural areas.

STUDY AREA

The study was conducted in southern Kane County and northern Kendall County, Illinois. The release site was the Lone Grove Forest Preserve (LGFP) in Kane County (80°33′45″E, 41°51′3″N). The 47.3-ha preserve included about 33.3 ha of mature upland forest in 2 blocks on either side of a paved road, 2 ha of old field undergoing succession, and 12 ha of open, mesic grassland bissected by a small creek.

The area surrounding LGFP was dominated by row-crop agriculture, primarily corn and soybeans. Agricultural fields were occasionally dissected by creeks or drainage ditches. A few isolated woodlots were scattered throughout the region. Most woodlots were small (<10 ha) and appeared as islands in the agricultural matrix. The only other trees in this landscape were associated with farmsteads, residential areas, and occasionally along creeks.

METHODS

We studied 3 treatment groups in each year: resident raccoons were livetrapped in LGFP, translocated rural raccoons were livetrapped in another wooded area (Max McGraw Wildlife Foundation, Dundee, Illinois, USA), and translocated urban raccoons were captured by licensed animal control agents in response to nuisance wildlife complaints (from Kane, DuPage, and Cook counties, Illinois, USA). In 1993, we captured and radiocollared resident raccoons from 30 August to 1 October, translocated rural raccoons from 2 to 29 September, and translocated urban raccoons from 6 to 29 September. In 1994, we captured and radiocollared resident raccoons from 28 July to 1 August, translocated rural raccoons from 3 to 11 August, and translocated urban raccoons from 14 August to 23 September. We followed raccoon movements from 1 September to 15 November 1993, and 28 July to 12 November 1994. We conducted our study in the fall of each year because fall is when the greatest numbers of nuisance raccoon complaints occur.

We captured raccoons in box traps, sedated them with Telazol, then weighed, measured, and ear-tagged them for individual identification. We fitted approximately equal numbers of adult males and females in each treatment group with radiocollars (Advanced Telemetry Systems, Isanti, Minnesota, USA). Radiocollars were equipped with a mortality switch and weighed 120–126 g. We held animals in their traps until they recovered from the anaesthetic, and we then released them at LGFP. We released all translocated animals at a parking area on the north side of LGFP because this was the standard practice of many nuisance animal control agents. We released resident raccoons either at their capture site or at the parking area if the capture site was less than about 500 m away, and we released raccoons within 24 hr of capture. The Laboratory Animal Care Advisory Committee of the University of Illinois, Urbana-Champaign approved all procedures for capture and handling of raccoons.

After release, we attempted to locate radiocollared raccoons daily until mid-November. We did not delay data collection for an acclimation period because some responses of interest (e.g., dispersal from the release site) typically occurred in the first few days after release. Azimuths were recorded via a vehicle-mounted, single-peak yagi antenna system. We recorded 4 azimuths/raccoon for each daily location from a set of fixed points corresponding to landscape features easily identifiable on maps and aerial photographs. Because our study area was generally covered with a grid system of county roads at 1.6-km (1-mile) intervals, we were able to confirm the locations by driving around the area until we had identified the particular landscape feature where the animal had denned. We then plotted daily locations of radiocollared raccoons on aerial photos. We determined linear distance from the release point, and we determined habitat classification for each location (woodlot, hedgerow, waterway, agricultural field, residential property). We used monthly checks from December through April in each year to monitor survival through the winter. Aerial searches (1 in 1993, 4 in 1994) were used to supplement ground searches.

We estimated survival functions via the Kaplan-Meier method (Kaplan and Meier 1958) for the period when daily locations were obtained for each treatment group. We estimated survival and its associated standard error with the Kaplan-Meier survivorship analysis program, Version 1.0 (Kulowiec 1988). We used log-rank tests (SPSS 1993) to compare differences among survival functions over time periods when we had complete data for all treatment groups (45 days in 1993, 50 days in 1994), but we present the survival functions for all data collected. We analyzed all data as days postrelease, because releases were staggered over 32 days in 1993 and 57 days in 1994. This staggering of release dates was because of the time required to capture equal numbers of adult male and female raccoons for each treatment group and waiting for suitable animals for the translocated urban sample to be captured in response to nuisance wildlife complaints. When the mortality switch was activated in a radiocollar, we located the dead raccoon (or the radiocollar if it had been removed) and attempted to determine the cause of mortality. We tested for differences in overwinter survival among treatment groups via chi-squared tests of independence.

To examine dispersal, we first compared the numbers of raccoons in each treatment group that remained near (<1 km from) the release site. The numbers in each treatment group still denning <1 km from the release site at 50 days postrelease were compared via chi-square tests of independence. Second, we compared mean linear distances from the release site for individuals that survived to the last 2 weeks of tracking in each treatment group in each year. Individuals that died or were lost before the last 2 weeks of tracking were not included in the statistical analysis. We used Kruskal-Wallis tests to examine differences among treatment groups because variances were not equal among groups. When mean linear distances differed ($P < 0.05$) among treatment groups, we used Mann-Whitney *U*-tests (SPSS 1993) to conduct pairwise comparisons between groups.

We classified daily den locations as occurring in 1 of 5 general habitat types: wooded areas, hedgerows, creeks or ditches, crop fields, or residential areas (farmsteads and other human habitations). Habitat use at this scale was easily determined in the field when we recorded azimuths, given the open landscape and grid-like system of rural roads. Because only some individuals, particularly resident raccoons, were located consistently, we quantified habitat use as frequency distributions for comparative purposes. A frequency distribution of den sites in each habitat was first calculated for each individual. Frequencies in each habitat type were then summed over all individuals in each treatment group for each year, and overall frequency distributions were recalculated from these summed values. Thus, each individual contributed equally to the final frequency distribution for each treatment group in each year.

RESULTS

We radiocollared and tracked 76 raccoons: 31 (15 M, 16 F) in 1993 and 45 (23 M, 22 F) in 1994. Sample size in each treatment group was equal within each year, with the exception that we radiocollared 11 raccoons in the translocated rural group in 1993 (instead of 10). We monitored radiocollared animals for 45–77 days in 1993 and 50–107 days in 1994.

Survival functions did not differ between males and females in either year (1993: $\chi^2_1 = 0.75$, $P = 0.39$; 1994: $\chi^2_1 = 1.38$, $P = 0.24$); therefore, we pooled sexes for further analysis. We analyzed each year separately because we tracked raccoons for >1 month longer in 1994 than in 1993.

Survival functions did not differ among the 3 treatment groups (1993: $\chi^2_2 = 0.18$, $P = 0.91$; 1994: $\chi^2_2 = 0.66$, $P = 0.72$; Fig. 1). We also did not detect differences in survival estimates among treatment groups in either year (mean estimate at 45 days postrelease for 1993: resident = 0.70 [95% CI = 0.27], translocated rural = 0.79 [95% CI = 0.27], translocated urban = 0.78 [95% CI = 0.31]; mean estimate at 50 days postrelease for 1994: resident = 0.93 [95% CI = 0.12], translocated rural = 0.85 [95% CI = 0.20], translocated urban = 0.87 [95% CI = 0.18]). Fourteen known deaths occurred during the daily radiotracking periods in 1993 and

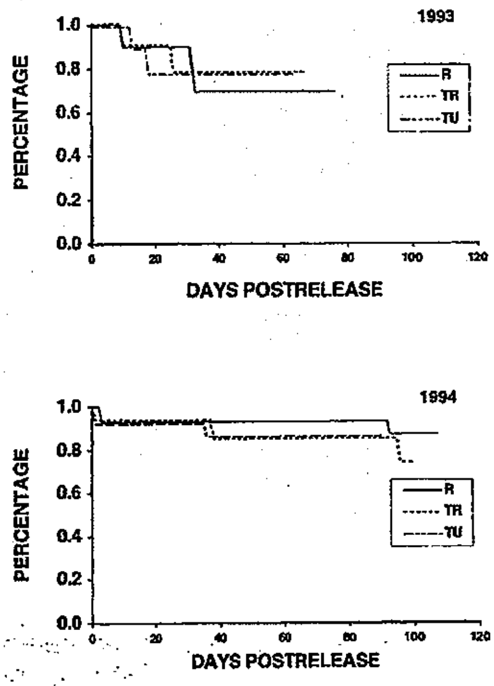


Fig. 1. Kaplan-Meier survival functions, estimated from days postrelease, for radiocollared raccoons trapped and released at the Lone Grove Forest Preserve, Kane County, Illinois (resident = R), trapped in another wooded area and translocated to the forest preserve (translocated rural = TR), and trapped as nuisance wildlife in suburban Chicago and translocated to the forest preserve (translocated urban = TU). Daily tracking was conducted from 30 August to 15 November in 1993 and from 28 July to 12 November in 1994. Curves are of different lengths because some groups were tracked for longer intervals than others.

1994: 5 raccoons were killed by vehicles, 4 were apparently killed by local homeowners (3 radiocollars were found discarded, 1 raccoon was found shot), 1 raccoon was trapped and killed as nuisance wildlife on private property, and 4 died of unknown causes (death occurred in tree cavities or in ground burrows, hence carcasses were not recovered for necropsy). Mortalities were distributed similarly among all 3 treatment groups (Table 1). In addition, 10 radiocollared animals were lost during the daily tracking periods, and their fates were unknown.

Of 19 radiocollared raccoons known alive at the end of the tracking period in 1993, 14 survived the winter, 3 died between November and April, and 2 disappeared (fates unknown). Of 33 radiocollared raccoons known alive at the end of the tracking period in 1994, 23 survived the winter, 5 died between November and April, and 5 disappeared (Table 1). Pooling data from both years, the numbers of raccoons that survived the winter, died during the winter, or were lost during the winter were not different among treatment groups ($\chi^2_4 = 3.86$, $P = 0.43$). Excluding lost animals from the analysis did not affect the results ($\chi^2_2 = 2.17$, $P = 0.34$).

Resident raccoons denned in or near LGFP during the radiotracking periods in both years, with the exception of 1 male in 1993 and 1 female in 1994 (the latter wandered widely but returned periodically until killed on the road about 1 month postrelease). In contrast, almost all translocated raccoons dispersed from the release site (Fig. 2). In 1993, only 2 of 21 translocated raccoons were still denning <1 km from the release site at the end of the tracking period, and most had left LGFP within 1–2 days postrelease. In 1994, only 4 of 30 translocated raccoons were still denning <1 km from the release site at the end of the tracking period. Pooling data from both years and excluding an-

Table 1. Fates of radiocollared raccoons trapped and released at the Lone Grove Forest Preserve, Kane County, Illinois (resident), trapped in another wooded area and translocated to the forest preserve (translocated rural), and trapped as nuisance wildlife in suburban Chicago and translocated to the forest preserve (translocated urban), 1993–94.

	1993			1994		
Treatment group	Survived	Died	Disappeared[a]	Survived	Died	Disappeared
Daily tracking period[b]						
Resident	7	3		13	2	
Translocated rural	7	2	2	8	3	4
Translocated urban	5	2	3	12	2	1
Overwinter[c]						
Resident	5	1	1	11	2	
Translocated rural	5	1	1	4	2	2
Translocated urban	4	1		8	1	3

[a] Individuals could no longer be located, no mortality signal detected.
[b] 1993: 30 August to 15 November; 1994: 28 July to 12 November.
[c] November to April.

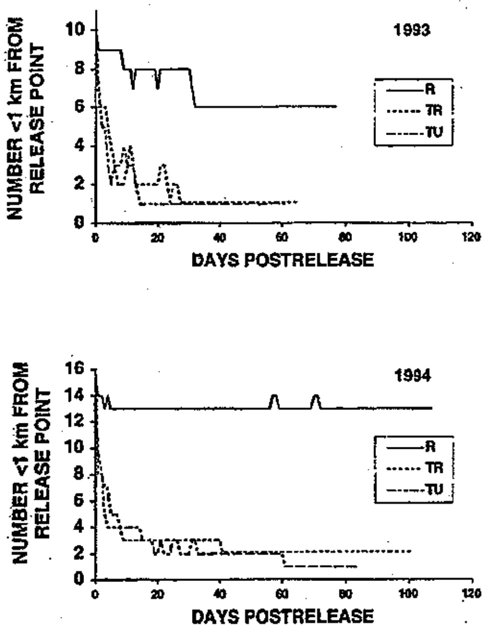


Fig. 2. Numbers of radiocollared raccoons trapped and released at the Lone Grove Forest Preserve, Kane County, Illinois (resident = R), trapped in another wooded area and translocated to the forest preserve (translocated rural = TR), and trapped as nuisance wildlife in suburban Chicago and translocated to the forest preserve (translocated urban = TU) that denned <1 km from the release site over time (days postrelease), 1993–94. Declines include losses due to both dispersal and mortality. Curves are of different lengths because some groups were tracked for longer intervals than others.

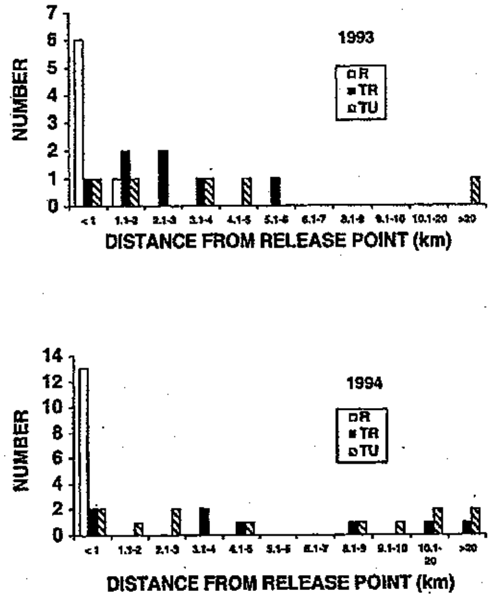


Fig. 3. Distances from the release site for radiocollared raccoons trapped and released at the Lone Grove Forest Preserve, Kane County, Illinois (resident = R), trapped in another wooded area and translocated to the forest preserve (translocated rural = TR), and trapped as nuisance wildlife in suburban Chicago and translocated to the forest preserve (translocated urban = TU) and known alive at the end of the period of daily radiotracking, 1993–94.

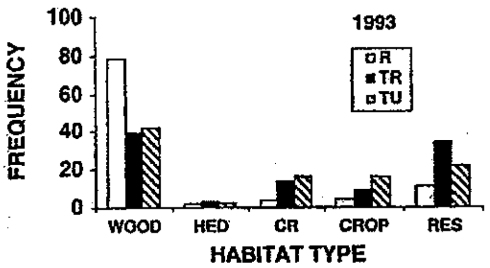
imals that died or were lost, 18 of 20 resident raccoons, 3 of 16 translocated rural raccoons, and 3 of 17 translocated urban raccoons were still denning <1 km from the release site at 50 days postrelease ($\chi^2_2 = 25.93$, $P < 0.001$). Differences in residency among the treatment groups were underestimated by this analysis because 4 of the raccoons in the resident sample that died before 50 days postrelease had consistently denned in the LGFP during the period they were monitored prior to their deaths.

Whereas most of the resident raccoons remained near the release site, many of the translocated rural and translocated urban raccoons dispersed considerable distances (Fig. 3). Three translocated raccoons were located 24, 25, and 60 km from the release site during aerial searches. The mean distance from the release site, excluding known mortalities, at the end of the period of daily tracking each year differed among treatment groups (1993: $\chi^2_2 = 7.78$, $P = 0.02$; 1994: $\chi^2_2 = 18.95$, $P < 0.001$). In both years, distances from the release site for resident raccoons (1993: 731 ± 424 m; 1994: 391 ± 125 m) were less than those for both translocated rural (1993: 3796 ± 2852 m; 1994: 9092 ± 8013 m) and translocated urban raccoons (1993: 7218 ± 8055 m; 1994: 7870 ± 6962 m; $Ps < 0.05$), whereas translocated groups did not differ in either year ($Ps > 0.05$).

Resident raccoons denned most frequently in wooded areas (i.e., LGFP; Fig. 4). Translocated raccoons frequently denned near human residences as well as in wooded areas, and they often denned in agricultural fields and along waterways.

DISCUSSION

In both years of our study, about 75–80% of the translocated raccoons survived until ≥2 months postrelease (Fig. 1), which did not differ from survival estimates for raccoons trapped at the release site. The survival of a translocated individual probably depends on a variety of factors including condition and health of the translocated animal, population density at the release site, area and quality of habitat at the release



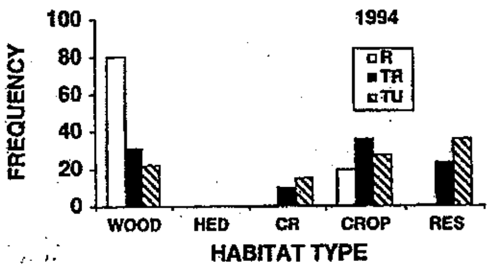


Fig. 4. Frequency distributions of habitats used for den sites during 1993–94 by radiocollared raccoons trapped and released at the Lone Grove Forest Preserve, Kane County, Illinois (resident = R), trapped in another wooded area and translocated to the forest preserve (translocated rural = TR), and trapped as nuisance wildlife in suburban Chicago and translocated to the forest preserve (translocated urban = TU), weighted so that each individual raccoon contributes equally. WOOD = woodlot or forest preserve; HED = hedgerow or treeline; CR = creek or drainage ditch; CROP = agricultural field; RES = farmstead, house, or other human construction.

site, and time of year. Frampton and Webb (1973) suggested translocations were most likely to succeed when population density at the release site was low. Raccoons were abundant in the area around LGFP; all 15 adult raccoons radiocollared in 1994 were captured in LGFP in only 5 nights (70 trapnights), and an attempt to identify resident raccoons in 1993 via a mark–recapture protocol resulted in 48 raccoons receiving ear tags in LGFP over a 45-day period in August–September. High survival rates of translocated raccoons in our study are probably attributable to benign weather conditions during autumn and abundant food and cover provided by agricultural crops.

The 10 "lost" raccoons in our study were treated as censored data in the calculation of survival function, and we assume they dispersed beyond the study area. Alternatively, their transmitters may have failed, but all 10 failures occurring in the translocated groups and none in the resident group is improbable. Only 3 raccoons were never relocated following release; 7 of the 10 "lost" raccoons were located for periods of 17–62 days postrelease, and all had dispersed from LGFP and moved extensively in the surrounding area prior to their last being located. Lost raccoons might have been resting in places such as culverts or barns where their signals were blocked. Finally, some of the lost raccoons could have been poached or killed as nuisance wildlife and their radiocollars disabled or discarded in places where signals would not be detected.

Rosatte and MacInnes (1989) reported that about 50% of 24 radiocollared raccoons transported from urban Toronto, Ontario, Canada, to a rural release site in the autumn died within 3 months of translocation. Wright (1977) also reported >57% mortality within 74 days of translocation for 28 radiocollared raccoons transported from Florida to Kentucky in the spring. In contrast, Taylor and Pelton (1979) found that 64% of 14 radiocollared raccoons translocated from bottomland forest to an upland forest preserve in the spring in Tennessee survived and established new home ranges, and Frampton and Webb (1973) detected only 20% mortalities for 10 radiocollared raccoons translocated from Coastal Plain to Upper Piedmont habitat in the spring in South Carolina, although the latter study had many technical difficulties. Thus, our survival estimates were higher than those of most other studies, but differences among habitats and timing of releases make comparisons difficult. Furthermore, none of the previous studies compared survival of translocated raccoons to that of a resident, control group.

Because we did not recapture radiocollared raccoons, we did not know the physical condition of animals at the beginning of winter. Overwinter survival rates were similar, however, among the 3 treatment groups. Severe winter weather did not begin until about 3 months after translocations were conducted, and translocated raccoons still being monitored at the end of the autumn radiotracking period had typically established new home ranges and den sites by that time.

In contrast to survival rates, dispersal patterns differed considerably between resident and translocated raccoons. Most of the resident sample remained in the vicinity of LGFP; with few exceptions, the linear distance between successive den sites averaged <400 m. Both translocated rural and translocated urban raccoons typically dispersed from the release site within hours to a few days. Dispersal could be a consequence of competition for den sites or other resources with resident animals at the release site, or due to disorientation and attempted homing by the released animals. The probability that a translocated raccoon would remain at the release site and the total distance dispersed from the release site by the end of the tracking period did not differ between raccoons translocated from urban to rural or from rural to rural environments.

The 24 translocated urban raccoons tracked by Rosatte and MacInnes (1989) similarly dispersed from their rural release sites but generally settled within 10–20 km of the release site. Other studies have reported some long-range movements by translocated raccoons: Wright (1977) reported mean dispersal distances of 27.5 km for 28 radiocollared and 25.7 km for 13 recovered (of 1,750 ear-tagged) raccoons moved from Florida to Kentucky, and Tabatabai and Kennedy (1989) reported a mean dispersal distance of 32.4 km for 25 recovered (of 450 ear-tagged) raccoons moved from urban to rural areas in Tennessee. The latter 2 studies detected maximum dispersal distances of 107 km and 295 km, respectively, from release sites for recovered, ear-tagged animals.

Because most of the translocated raccoons did not stay at the release site and sometimes dispersed considerable distances, translocated animals could add to nuisance wildlife problems for rural human residents or increase the spread of disease during zoonotic outbreaks. Raccoons exhibit high incidences of exposure to various wildlife diseases (e.g., Krebs et al. 1995, Centers for Disease Control 1997), and the risk of translocating diseases or parasites along with nuisance animals is an important consideration (Cunningham 1996). Hence, because some translocated animals disperse for long distances after translocation, the rate of spread of zoonotic disease could be accelerated considerably. Indeed, many translocated raccoons denned near human residences or on residential property, including backyards, barns, houses, and even a gas station. At least 4 translocated raccoons were captured or killed by local human residents because of nuisance behavior (1 during the tracking period, 3 during the winter of 1994).

Further research on the effects of translocating raccoons is warranted because of the potential consequences for other wildlife species. Raccoons are important nest predators on songbirds (Whelan et al. 1994, Donovan et al. 1997), game birds (Miller and Leopold 1992), waterfowl (Urban 1970, Duebbert and Kantrud 1974, Greenwood 1981, Jobin and Picman 1997), and other wildlife (e.g., turtles; Christens and Bider 1987, Congdon et al. 1987). Translocated raccoons also may compound the intense predation pressure already experienced by some wildlife in fragmented habitats. In some areas, existing management practices such as waterfowl management (Urban 1970, Fritzell 1978, Sargeant et al. 1993) near a release site could be jeopardized by an infusion of translocated raccoons. Finally, increased competition for food and den sites, or disruption of social organization, could negatively affect resident raccoons.

MANAGEMENT IMPLICATIONS

Our study shows that survival rates of translocated raccoons can be as high as those of resident animals. This finding supports the view of some that translocation is a humane method for handling nuisance wildlife problems (Diehl 1988). However, large numbers of raccoons translocated into an area could increase competition for resources with resident raccoons, predation pressure on other wildlife, and nuisance wildlife problems for human residents near release sites. Records from licensed animal controllers in Kane County show that ≥84 raccoons were released in LGFP in 1993 prior to our study, and 823 more were released at other sites within the county. Raccoons are abundant in most of the Midwest, and there are few, if any, places to release a translocated raccoon where there is not already a substantial population of other raccoons or people. Further, most nature preserves around urban or suburban areas are relatively small and are unlikely to accommodate the thousands of animals handled by animal control professionals each year. Even when a translocated raccoon survives, however, our study indicates it rarely stays at the release site. When the risk of facilitating the spread of disease during zoonotic outbreaks also is considered, translocation of nuisance wildlife becomes a less attractive option.

Alternatives to translocation for solving nuisance wildlife problems also have drawbacks. The simplest alternative is euthanasia, but it is the most controversial. The use of reproductive inhibitors (Howard 1967) or surgical sterilization (Bojrab et al. 1983) is costly and labor intensive. Physically excluding nuisance wildlife from private property would be the most humane solution but may be difficult and ineffective. Thus, the negative effects of translocating large numbers of animals on wildlife and human residents of rural areas near release sites must be weighed against the negative public opinion and ethical considerations concerning euthanasia or sterilization when determining policy for the disposition of nuisance wildlife.

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